Baskar, P.
10/726692

Seq IDs

FILE 'REGISTRY' ENTERED AT 08:52:32 ON 23 APR 2004
L1 313 SEA ABB=ON PLU=ON LKEKAK|GHEGCRSGEAEGS|SHEGCRSGEAEGS/SQSP

FILE 'HCAPLUS' ENTERED AT 08:53:40 ON 23 APR 2004
L2 209 SEA ABB=ON PLU=ON L1
L3 24 SEA ABB=ON PLU=ON L2 AND RECOMBINAN?
L4 19 SEA ABB=ON PLU=ON L3 AND (DIAGNOS? OR DETERM? OR DET## OR SCREEN? OR DETECT?)

L4 ANSWER 1 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 22 Feb 2004
ACCESSION NUMBER: 2004:142686 HCAPLUS
DOCUMENT NUMBER: 140:176312
TITLE: Novel 12 human genes and 32252 and uses therefor in modulating cellular growth and function in cancer therapy
INVENTOR(S): Meyers, Rachel E.; Williamson, Mark J.; Kapeller-Libermann, Rosana; Macbeth, Kyle J.; Hunter, John Joseph; Rudolph-Owen, Laura A.; Bandaru, Rajasekhar; Tsai, Fong-Ying
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 149 pp., Cont.-in-part of U.S. Ser. No. 251,507.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004033509	A1	20040219	US 2003-377097	20030228
US 2002068698	A1	20020606	US 2001-910150	20010718
PRIORITY APPLN. INFO.:			US 2000-219028P P	20000718
			US 2001-910150 B2	20010718
			US 2002-251507 A2	20020920

AB The invention provides cDNA and protein sequences for 12 novel human genes, designated 13237, 18480, 2245, 16228, 7677, 26320, 46619, 33166, 16836, 46867, 21617, 55562, 39228, 62088, 46745, 23155, 21657, 42755, 32229, 22325, 46863 and 32252 nucleic acid mols. The invention also provides antisense nucleic acid mols., **recombinant** expression vectors containing these genes, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which one of these genes has been introduced or disrupted. The invention still further provides their coded proteins, fusion proteins, antigenic peptides and antibodies. **Diagnostic** and therapeutic methods utilizing compns. of the invention are also provided.

IT **660003-26-9P**, Protein (human gene 32229)
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; novel 12 human genes and 32252, and uses therefor in modulating cellular growth and function in cancer

therapy)

L4 ANSWER 2 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 08 Jan 2004
ACCESSION NUMBER: 2004:12857 HCAPLUS
DOCUMENT NUMBER: 140:88747
TITLE: Nucleic acid and amino acid sequences relating to Moraxella catarrhalis for **diagnostics** and therapeutics
INVENTOR(S): Breton, Gary L.
PATENT ASSIGNEE(S): Genome Therapeutics Corporation, USA
SOURCE: U.S., 429 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6673910	B1	20040106	US 2000-540236	20000404
PRIORITY APPLN. INFO.:			US 1999-128416P P	19990408

AB The invention provides isolated polypeptide and nucleic acid sequences derived from Moraxella catarrhalis that are useful in **diagnosis** and therapy of pathol. conditions; antibodies against the polypeptides; and methods for the production of the polypeptides. The invention provides 1920 genomic nucleic acids corresponding to entire coding sequences of M. catarrhalis proteins and two naturally occurring plasmids in M. catarrhalis, including surface or secreted proteins or parts thereof, nucleic acids capable of binding mRNA from M. catarrhalis, proteins to block protein translation, and methods for producing M. catarrhalis proteins or parts thereof using peptide synthesis and **recombinant** DNA techniques. M. catarrhalis is known to be involved in otitis media and respiratory tract infections. Thus, the invention also provides methods for the **detection**, prevention, and treatment of pathol. conditions resulting from bacterial infection.

IT **642131-60-0**
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; nucleic acid and amino acid sequences relating to Moraxella catarrhalis for **diagnostics** and therapeutics)

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 09 Oct 2003
ACCESSION NUMBER: 2003:790302 HCAPLUS
DOCUMENT NUMBER: 140:284548
TITLE: Xenopus nucleosome assembly protein becomes tissue-restricted during development and can alter the expression of specific genes
AUTHOR(S): Steer, Wendy M.; Abu-Daya, Anita; Brickwood,

Sarah J.; Mumford, Katherine L.; Jordanaires, Niove; Mitchell, Julian; Robinson, Carl; Thorne, Alan W.; Guille, Matthew J.

CORPORATE SOURCE: Institute of Biomedical and Biomolecular Sciences, Genes and Development, University of Portsmouth, Portsmouth, PO1 2DY, UK

SOURCE: Mechanisms of Development (2003), 120(9), 1045-1057
CODEN: MEDVE6; ISSN: 0925-4773

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Nucleosome assembly proteins have been identified in all eukaryotic species investigated to date and their suggested roles include histone shuttle, histone acceptor during transcriptional chromatin remodelling, and cell cycle regulator. To examine the role of these proteins during early development we have isolated the cDNA encoding Xenopus NAP1L, raised an antibody against **recombinant** xNAP1L and examined the expression pattern of this mRNA and protein. Expression in adults is predominantly in ovaries. This maternal protein remains a major component of xNAP1L within the embryo until swimming tadpole stages. XNAP1L mRNA is initially throughout the embryo but by gastrula stages it is predominantly in the presumptive ectoderm. Later, mRNA is **detected** in the neural crest, neural tube, eyes, tailbud, and ventral blood islands. To test whether xNAP1L has a potential role in gene regulation we overexpressed this protein in animal pole explants and tested the effect on expression of a series of potential target genes. The mRNA encoding the transcription factor GATA-2 was markedly up-regulated by this overexpression. These data support a role for xNAP1L in tissue-restricted gene regulation.

IT **674875-34-4**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; nucleosome assembly protein sequence and distribution in frog in development)

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

ED Entered STN: 26 Sep 2003

ACCESSION NUMBER: 2003:757225 HCAPLUS

DOCUMENT NUMBER: 139:272071

TITLE: Gene disruption method GRACE (gene replacement and conditional expression) for identification of drug targets in Candida albicans and other diploid fungal pathogens

INVENTOR(S): Roemer, Terry; Jiang, Bo; Boone, Charles; Bussey, Howard; Ohlsen, Kari L.

PATENT ASSIGNEE(S): Elitra Pharmaceuticals, Inc., USA

SOURCE: U.S. Pat. Appl. Publ., 79 pp., Cont.-in-part of U.S. Ser. No. 792,024.
CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003180953	A1	20030925	US 2001-32585	20011220

PRIORITY APPLN. INFO.: US 2000-259128P P 20001229
US 2001-792024 A2 20010220
US 2001-314050P P 20010822

AB The present invention provides methods and compns. that enable the exptl. **determination** as to whether any gene in the genome of a diploid pathogenic organism is essential, and whether it is required for virulence or pathogenicity. The method designated GRACE (gene replacement and conditional expression) involve the construction of genetic mutants in which one allele of a specific gene is inactivated while the other allele of the gene is placed under conditional expression. The identification of essential genes and those genes critical to the development of virulent infections, provides a basis for the development of **screens** for new drugs against such pathogenic organisms. The present invention further provides 932 Candida albicans genes that are demonstrated to be essential and are potential targets for drug **screening**. The nucleotide sequence of the target genes can be used for various drug discovery purposes, such as expression of the **recombinant** protein, hybridization assay, and construction of nucleic acid arrays. The uses of proteins encoded by the essential genes, and genetically engineered cells comprising modified alleles of essential genes in various **screening** methods are also encompassed by the invention.

IT **604828-86-6**
RL: AGR (Agricultural use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; GRACE (gene replacement and controlled expression) and use of gene disruption methods for identification of drug targets in diploid fungal pathogens)

L4 ANSWER 5 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 18 Jun 2003
ACCESSION NUMBER: 2003:464737 HCAPLUS
DOCUMENT NUMBER: 139:2147
TITLE: Human colon and colon cancer-associated polynucleotides and polypeptides and their **diagnostic** and therapeutic applications
INVENTOR(S): Ruben, Steven M.; Barash, Steve C.; Birse, Charles E.; Rosen, Craig A.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 174 pp., Cont.-in-part of Appl. No. PCT/US00/26524.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

US 2003109690 A1 20030612 US 2002-106698 20020327
WO 2001022920 A2 20010405 WO 2000-US26524 20000928
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
US 2003109690 A1 20030612 US 2002-106698 20020327
PRIORITY APPLN. INFO.: US 1999-157137P P 19990929
US 1999-163280P P 19991103
WO 2000-US26524 A2 20000928
US 2002-106698 A 20020327

AB The present invention relates to 8354 novel colon- or colon cancer-related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "colon or colon cancer antigens," and the use of such colon or colon cancer antigens for **detecting** disorders of the colon, particularly the presence of colon cancer and colon cancer metastases. More specifically, isolated colon- or colon cancer-associated nucleic acid mols. are provided encoding novel colon- or colon cancer-associated polypeptides. Novel colon or colon cancer polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and **recombinant** and synthetic methods for producing human colon- or colon cancer-associated polynucleotides and/or polypeptides. The invention further relates to **diagnostic** and therapeutic methods useful for **diagnosing**, treating, preventing and/or prognosing disorders related to the colon, including colon cancer, and therapeutic methods for treating such disorders. The invention further relates to **screening** methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compns. for inhibiting the production and function of the polypeptides of the present invention. [This abstract record is one of two records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

IT **536052-37-6P**
RL: ADV (Adverse effect, including toxicity); BPN (Biosynthetic preparation); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; human colon and colon cancer-associated polynucleotides and polypeptides and their **diagnostic** and therapeutic applications)

L4 ANSWER 6 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 02 Aug 2002
ACCESSION NUMBER: 2002:572038 HCAPLUS
DOCUMENT NUMBER: 137:104826
TITLE: Identification of essential genes in prokaryotes and use of their antisense constructs in

TITLE: antibiotic **screening**
INVENTOR(S): Roemer, Terry; Jiang, Bo; Boone, Charles; Bussey, Howard; Ohlsen, Kari L.
PATENT ASSIGNEE(S): Elitra Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 167 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002053728	A2	20020711	WO 2001-XA49486	20011226

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 2000-259128P P 20001229
US 2001-792024 A 20010220
US 2001-314050P P 20010822

AB The present invention provides methods and compns. that enable the exptl. **determination** as to whether any gene in the genome of a diploid pathogenic organism is essential, and whether it is required for virulence or pathogenicity. The methods involve the construction of genetic mutants in which one allele of a specific gene is inactivated while the other allele of the gene is placed under conditional expression. The identification of essential genes and those genes critical to the development of virulent infections, provides a basis for the development of **screens** for new drugs against such pathogenic organisms. The present invention further provides Candida albicans genes that are demonstrated to be essential and are potential targets for drug **screening**. The nucleotide sequence of the target genes can be used for various drug discovery purposes, such as expression of the **recombinant** protein, hybridization assay and construction of nucleic acid arrays. The uses of proteins encoded by the essential genes, and genetically engineered cells comprising modified alleles of essential genes in various **screening** methods are also encompassed by the invention. The patent has clamid 7932 DNA and protein sequences, but the sequence information is not avaliable upon the time of this publication.

IT **443161-46-4**
RL: ARU (Analytical role, unclassified); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(amino acid sequence; Identification of essential genes in prokaryotes and use of their antisense constructs in antibiotic **screening**)

L4 ANSWER 7 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 03 May 2002
ACCESSION NUMBER: 2002:332629 HCAPLUS
DOCUMENT NUMBER: 136:352023
TITLE: Cloning, characterization and therapeutic use of human dehydrogenase isoenzymes
INVENTOR(S): Meyers, Rachel; Cook, William James; Williamson, Mark; Rudolph-Owen, Laura A.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 104 pp., Cont.-in-part of U.S. Ser. No. 634,955.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002052032	A1	20020502	US 2001-816760	20010323
US 6613555	B2	20030902		
US 6511834	B1	20030128	US 2000-634955	20000808
US 2002042371	A1	20020411	US 2001-838561	20010418
US 6627423	B2	20030930		
US 2003166200	A1	20030904	US 2002-172585	20020614
US 2004077010	A1	20040422	US 2003-664506	20030917

PRIORITY APPLN. INFO.:
US 2000-192002P P 20000324
US 2000-634955 A2 20000808
US 2001-816760 A2 20010323
US 2001-838561 A1 20010418

AB The invention provides isolated nucleic acids mols., designated DHDR, which encode novel DHDR-related dehydrogenase mols. The invention also provides antisense nucleic acid mols., **recombinant** expression vectors containing DHDR nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a DHDR gene has been introduced or disrupted. The invention still further provides isolated DHDR proteins, fusion proteins, antigenic peptides and anti-DHDR antibodies. The cDNA sequences and predicted amino acid sequences of human DHDR-1 (clone Fbh32142), DHDR-2 (clone Fbh21481), DHDR-3 (clone Fbh25964), and DHDR-4 (clone Fbh21686) are provided. The expression levels of human DHDR isoenzymes in various human cell types and tissues are disclosed. **Diagnostic** methods utilizing compns. of the invention are provided.

IT **420277-73-2DP**, subfragments and variants are claimed
RL: ANT (Analyte); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; cloning, characterization and therapeutic use of human dehydrogenase isoenzymes)

L4 ANSWER 8 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 26 Apr 2002
ACCESSION NUMBER: 2002:315079 HCAPLUS

DOCUMENT NUMBER: 136:336297
TITLE: Protein and cDNA sequences of a novel human acyl-CoA dehydrogenase sequence homolog and **diagnostic** and therapeutic uses thereof
INVENTOR(S): Kapeller-Libermann, Rosana; Hunter, John J.; Rudolph-Owen, Laura A.
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 115 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002033061	A2	20020425	WO 2001-US46720	20011022
WO 2002033061	A3	20030227		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002028829	A5	20020429	AU 2002-28829	20011022
US 2003040474	A1	20030227	US 2001-999314	20011022
EP 1339835	A2	20030903	EP 2001-987799	20011022
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: US 2000-242211P P 20001020
WO 2001-US46720 W 20011022

AB The invention provides protein and cDNA sequences of a novel human protein, designated 32229, which has sequence homol. with acyl-CoA dehydrogenase members. The invention also provides antisense nucleic acid mols., **recombinant** expression vectors containing 32229 nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 32229 gene has been introduced or disrupted. The invention still further provides isolated 32229 proteins, fusion proteins, antigenic peptides and anti-32229 antibodies. **Diagnostic** methods utilizing compns. of the invention are also provided.

IT **416229-10-2P**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; protein and cDNA sequences of novel human acyl-CoA dehydrogenase sequence homolog and **diagnostic** and therapeutic uses thereof)

L4 ANSWER 9 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

ED Entered STN: 12 Apr 2002
ACCESSION NUMBER: 2002:276516 HCAPLUS
DOCUMENT NUMBER: 136:305208
TITLE: Protein and cDNA sequences of novel dehydrogenase sequence homologs and uses thereof
INVENTOR(S): Meyers, Rachel; Cook, William James; Williamson, Mark; Rudolph-Owen, Laura A.; Gimeno, Ruth
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 114 pp., Cont.-in-part of U.S. Ser. No. 816,760.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002042371	A1	20020411	US 2001-838561	20010418
US 6627423	B2	20030930		
US 6511834	B1	20030128	US 2000-634955	20000808
US 2002052032	A1	20020502	US 2001-816760	20010323
US 6613555	B2	20030902		
US 2004077010	A1	20040422	US 2003-664506	20030917

PRIORITY APPLN. INFO.:
US 2000-192002P P 20000324
US 2000-634955 A2 20000808
US 2001-816760 A2 20010323
US 2001-838561 A1 20010418

AB The invention provides protein and cDNA sequences of four novel human proteins and one mouse protein, designated DHDR, which have sequence homol. with dehydrogenase family members. The invention also provides antisense nucleic acid mols., **recombinant** expression vectors containing DHDR nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a DHDR gene has been introduced or disrupted. The invention still further provides isolated DHDR proteins, fusion proteins, antigenic peptides and anti-DHDR antibodies. **Diagnostic** methods utilizing compns. of the invention are also provided.

IT **410115-99-0P**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; protein and cDNA sequences of novel dehydrogenase sequence homologs and uses thereof)

IT **410117-83-8**
RL: PRP (Properties)
(unclaimed protein sequence; protein and cDNA sequences of novel dehydrogenase sequence homologs and uses thereof)

L4 ANSWER 10 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 21 Dec 2001
ACCESSION NUMBER: 2001:923966 HCAPLUS
DOCUMENT NUMBER: 136:52364
TITLE: Polynucleotides differentially expressed in

colon cancers, their encoded polypeptides, **diagnostic** and therapeutic uses

INVENTOR(S): Kennedy, Giulia C.; Kang, Sanmao; Reinhard, Christoph; Jefferson, Anne Bennet

PATENT ASSIGNEE(S): Chiron Corporation, USA

SOURCE: PCT Int. Appl., 135 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001096523	A2	20011220	WO 2001-US19313	20010615
WO 2001096523	A3	20030710		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001069867	A5	20011224	AU 2001-69867	20010615
US 2003008284	A1	20030109	US 2001-883152	20010615
EP 1370684	A2	20031217	EP 2001-948414	20010615
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
JP 2004503238	T2	20040205	JP 2002-510643	20010615

PRIORITY APPLN. INFO.: US 2000-211835P P 20000615
WO 2001-US19313 W 20010615

AB The present invention is based on the discovery of polynucleotides that represent genes that are differentially expressed in colon cancer, e.g., adenomatous polyp, colorectal carcinoma, high metastatic potential colon tumor and metastatic colon cancer. The invention features methods of identifying cells affected by such colon diseases by **detection** of a gene product encoded by such differentially expressed genes, as well as method of modulating expression of such gene products to effect therapy (e.g., to decrease growth and/or affect abnormal characteristics of cancerous or displastic colon cells). CDNA libraries from cell lines and tissue sources were grouped into clusters of signature sequences by hybridization and computational anal. Clones which correspond to genes that are differentially expressed in tissues from colon cancer patients were identified by comparing the cDNA library clusters. Overexpression of the cloned cDNAs was measured with tissue samples and colon epithelial cells from patients with colon cancer by RT-PCR. Expression of clone SK2 was further analyzed in different cancer cell lines. Some antisense oligonucleotides for genes corresponding to the isolated clones lowered mRNA expression, inhibited cell proliferation, reduced colony formation in soft agar, and affected apoptosis as measured by cellular assays. Differential

gene expression was also analyzed with cDNA microarrays using labeled cDNA probes prepared from total RNA from normal and cancerous cells.

IT **382665-85-2**, Protein (human clone XD4)
RL: ANT (Analyte); DGN (Diagnostic use); PRP (Properties); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(amino acid sequence; polynucleotides differentially expressed in colon cancers, their encoded polypeptides, **diagnostic** and therapeutic uses)

L4 ANSWER 11 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 07 Dec 2001
ACCESSION NUMBER: 2001:886497 HCAPLUS
DOCUMENT NUMBER: 136:33913
TITLE: Anthocyanin 3'-O-glucosyltransferase from Gentiana triflora, Senecio cruentus, and Clitoria ternatea by mRNA differential display for plant forms regarding anthocyanin
INVENTOR(S): Mizutani, Masako; Sakakibara, Keiko; Tanaka, Yoshikazu; Kusumi, Takaaki; Ono, Eiichiro
PATENT ASSIGNEE(S): International Flower Developments Proprietary Limited, Australia
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001092509	A1	20011206	WO 2001-JP4675	20010601
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001060699	A5	20011211	AU 2001-60699	20010601
EP 1291418	A1	20030312	EP 2001-934509	20010601
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: JP 2000-170436 A 20000602
WO 2001-JP4675 W 20010601

AB UDP-glucose:anthocyanin 3'-O-glucosyltransferase (3-GT) that catalyzes the transfer of glycosyl group to the 3'-position of anthocyanin, cDNAs, **recombinant** expression, and use in modifying flower color in plants, are disclosed. An enzyme catalyzing the synthesis of delphinidin 3,3',5-triglycoside from UDP-glucose and delphinidin 3,5-diglucoside was isolated from Gentiana triflora and its partial amino acid sequences were

determined A cDNA coding for the enzyme was cloned and expressed in yeast, E. coli, and Petunia. The enzyme activity was characterized to show the reactivity toward delphinidin 3-glycosyl-5-caffeoylglucoside as well. CDNA was also cloned from cineraria and Clitoria ternatea.

IT **379739-09-0P**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); CAT (Catalyst use); PRP (Properties); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; anthocyanin 3'-O-glucosyltransferase from Gentiana triflora, Senecio cruentus, and Clitoria ternatea by mRNA differential display for plant forms regarding anthocyanin)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 12 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 07 Oct 2001
ACCESSION NUMBER: 2001:730945 HCAPLUS
DOCUMENT NUMBER: 135:285001
TITLE: Protein and cDNA sequences of a novel human dehydrogenase sequence homolog and uses thereof
INVENTOR(S): Meyers, Rachel; Cook, William James; Williamson, Mark; Rudolph-Owen, Laura A.
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 165 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001072976	A2	20011004	WO 2001-US9613	20010323
WO 2001072976	A3	20020906		
WO 2001072976	C2	20030403		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6511834	B1	20030128	US 2000-634955	20000808
EP 1268809	A2	20030102	EP 2001-920767	20010323
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003166200	A1	20030904	US 2002-172585	20020614

PRIORITY APPLN. INFO.:
US 2000-192002P P 20000324
US 2000-634955 A 20000808
WO 2001-US9613 W 20010323

AB The invention provides isolated nucleic acids mols., designated DHDR nucleic acid mols., which encode novel DHDR-related dehydrogenase mols. The invention also provides antisense nucleic acid mols., **recombinant** expression vectors containing DHDR nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a DHDR gene has been introduced or disrupted. The invention still further provides isolated DHDR proteins, fusion proteins, antigenic peptides and anti-DHDR antibodies. **Diagnostic** methods utilizing compns. of the invention are also provided.

IT **364638-73-3**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(amino acid sequence; protein and cDNA sequences of novel human dehydrogenase sequence homolog and uses thereof)

IT **364639-16-7**
RL: PRP (Properties)
(unclaimed sequence; protein and cDNA sequences of a novel human dehydrogenase sequence homolog and uses thereof)

L4 ANSWER 13 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 03 Aug 2001
ACCESSION NUMBER: 2001:565201 HCAPLUS
DOCUMENT NUMBER: 135:163399
TITLE: Human nucleic acids and their encoded proteins and antibodies
INVENTOR(S): Rosen, Craig A.; Barash, Steven C.; Ruben, Steven M.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: PCT Int. Appl., 980 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 93
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001055322	A2	20010802	WO 2001-US1341	20010117
WO 2001055322	A3	20020704		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001041413	A5	20010807	AU 2001-41413	20010117
AU 2001041416	A5	20010807	AU 2001-41416	20010117
AU 2001041417	A5	20010807	AU 2001-41417	20010117
AU 2001050770	A5	20010807	AU 2001-50770	20010117

AU 2001055162	A5	20010807	AU 2001-55162	20010117
US 2002042096	A1	20020411	US 2001-764887	20010117
US 2002077270	A1	20020620	US 2001-764848	20010117
US 2002086811	A1	20020704	US 2001-764861	20010117
US 2003171252	A9	20030911		
US 2002086820	A1	20020704	US 2001-764862	20010117
US 2003092611	A9	20030515		
US 2002086821	A1	20020704	US 2001-764881	20010117
US 2003125246	A9	20030703		
US 2002086822	A1	20020704	US 2001-764886	20010117
US 2003139327	A9	20030724		
US 2002086823	A1	20020704	US 2001-764889	20010117
US 2002086330	A1	20020704	US 2001-764893	20010117
US 2002090615	A1	20020711	US 2001-764878	20010117
US 2002090674	A1	20020711	US 2001-764903	20010117
US 2002094953	A1	20020718	US 2001-764860	20010117
US 2002102638	A1	20020801	US 2001-764846	20010117
US 2002119919	A1	20020829	US 2001-764855	20010117
US 2002132767	A1	20020919	US 2001-764847	20010117
US 2002147140	A1	20021010	US 2001-764877	20010117
US 2002151479	A1	20021017	US 2001-764873	20010117
US 2002161208	A1	20021031	US 2001-764884	20010117
EP 1254147	A2	20021106	EP 2001-928288	20010117
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2002164685	A1	20021107	US 2001-764857	20010117
US 2002173454	A1	20021121	US 2001-764904	20010117
US 2003044890	A1	20030306	US 2001-764876	20010117
US 2003050231	A1	20030313	US 2001-764872	20010117
AU 2001052878	A5	20010807	AU 2001-52878	20010129
AU 2001043137	A5	20010814	AU 2001-43137	20010205
AU 2001041411	A5	20010820	AU 2001-41411	20010208
US 2002045230	A1	20020418	US 2001-908711	20010720
US 2003013649	A1	20030116	US 2001-989442	20011121
US 2003054420	A1	20030320	US 2002-72349	20020211
US 2003044904	A1	20030306	US 2002-73865	20020214
US 2003044905	A1	20030306	US 2002-73979	20020214
US 2003077703	A1	20030424	US 2002-73912	20020214
US 2003077602	A1	20030424	US 2002-73961	20020214
US 2003077704	A1	20030424	US 2002-74095	20020214
US 2003092102	A1	20030515	US 2002-74045	20020214
US 2003096346	A1	20030522	US 2002-73885	20020214
US 2003039993	A1	20030227	US 2002-79900	20020222
US 2003044907	A1	20030306	US 2002-80110	20020222
US 2003054368	A1	20030320	US 2002-79854	20020222
US 2003039994	A1	20030227	US 2002-91526	20020307
US 2003049650	A1	20030313	US 2002-91483	20020307
US 2003049703	A1	20030313	US 2002-91548	20020307
US 2003054373	A1	20030320	US 2002-91572	20020307
US 2003054375	A1	20030320	US 2002-92154	20020307
US 2003059908	A1	20030327	US 2002-91504	20020307
US 2003068627	A1	20030410	US 2002-91458	20020307
US 2003077606	A1	20030424	US 2002-91438	20020307
US 2003082681	A1	20030501	US 2002-91391	20020307
US 2003054377	A1	20030320	US 2002-102627	20020322
US 2003082758	A1	20030501	US 2002-103313	20020322

Searcher : Shears 571-272-2528

US 2003054379	A1	20030320	US 2002-116016		20020405
US 2003092615	A1	20030515	US 2002-115928		20020405
US 2003059875	A1	20030327	US 2002-125540		20020419
PRIORITY APPLN. INFO.:			US 2000-179065P	P	20000131
			US 2000-180628P	P	20000204
			US 2000-184664P	P	20000224
			US 2000-186350P	P	20000302
			US 2000-189874P	P	20000316
			US 2000-190076P	P	20000317
			US 2000-198123P	P	20000418
			US 2000-205515P	P	20000519
			US 2000-209467P	P	20000607
			US 2000-214886P	P	20000628
			US 2000-215135P	P	20000630
			US 2000-216647P	P	20000707
			US 2000-216880P	P	20000707
			US 2000-217487P	P	20000711
			US 2000-217496P	P	20000711
			US 2000-218290P	P	20000714
			US 2000-220963P	P	20000726
			US 2000-220964P	P	20000726
			US 2000-224518P	P	20000814
			US 2000-224519P	P	20000814
			US 2000-225213P	P	20000814
			US 2000-225214P	P	20000814
			US 2000-225266P	P	20000814
			US 2000-225267P	P	20000814
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			US 2000-225270P	P	20000814
			US 2000-225447P	P	20000814
			US 2000-225757P	P	20000814
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			US 2000-225759P	P	20000814
			US 2000-226279P	P	20000818
			US 2000-226681P	P	20000822
			US 2000-226868P	P	20000822
			US 2000-227182P	P	20000822
			US 2000-227009P	P	20000823
			US 2000-228924P	P	20000830
			US 2000-229343P	P	20000901
			US 2000-229344P	P	20000901
			US 2000-229287P	P	20000901
			US 2000-229345P	P	20000901
			US 2000-229509P	P	20000905
			US 2000-229513P	P	20000905
			US 2000-231413P	P	20000908
			US 2000-232398P	P	20000914
			US 2000-234223P	P	20000921
			US 2000-234274P	P	20000921
			US 2000-234997P	P	20000925
			US 2000-235834P	P	20000927
			US 2000-236327P	P	20000929
			US 2000-236367P	P	20000929
			US 2000-236368P	P	20000929
			US 2000-236369P	P	20000929
			US 2000-236370P	P	20000929

Searcher : Shears 571-272-2528

US 2000-236802P	P	20001002
US 2000-237037P	P	20001002
US 2000-237039P	P	20001002
US 2000-237040P	P	20001002
US 2000-241221P	P	20001020
US 2000-241785P	P	20001020
US 2000-241786P	P	20001020
US 2000-241809P	P	20001020
US 2000-244617P	P	20001101
US 2000-246478P	P	20001108
US 2000-246523P	P	20001108
US 2000-246524P	P	20001108
US 2000-246609P	P	20001108
US 2000-246613P	P	20001108
US 2000-249207P	P	20001117
US 2000-249208P	P	20001117
US 2000-249210P	P	20001117
US 2000-249211P	P	20001117
US 2000-249212P	P	20001117
US 2000-249213P	P	20001117
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US 2000-249245P	P	20001117
US 2000-249297P	P	20001117
US 2000-249299P	P	20001117
US 2000-249300P	P	20001117
US 2000-250160P	P	20001201
US 2000-251856P	P	20001208
US 2000-251868P	P	20001208
US 2000-251869P	P	20001208
US 2000-251990P	P	20001208
US 2001-764847	B1	20010117
US 2001-764848	B1	20010117
US 2001-764850	B1	20010117
US 2001-764852	B1	20010117
US 2001-764853	A2	20010117
US 2001-764854	B1	20010117
US 2001-764855	B1	20010117
US 2001-764856	A2	20010117
US 2001-764857	B1	20010117
US 2001-764860	B1	20010117
US 2001-764861	A1	20010117
US 2001-764862	A1	20010117
US 2001-764863	B1	20010117
US 2001-764864	A2	20010117
US 2001-764866	B1	20010117
US 2001-764867	A2	20010117
US 2001-764868	A2	20010117
US 2001-764869	A2	20010117
US 2001-764870	A2	20010117
US 2001-764873	B1	20010117
US 2001-764874	A2	20010117
US 2001-764878	A1	20010117

Searcher : Shears 571-272-2528

US	2001-764879	B1 20010117
US	2001-764882	A2 20010117
US	2001-764885	B1 20010117
US	2001-764887	B1 20010117
US	2001-764888	A2 20010117
US	2001-764889	A1 20010117
US	2001-764891	A2 20010117
US	2001-764892	A2 20010117
US	2001-764893	B1 20010117
US	2001-764896	A2 20010117
US	2001-764898	A2 20010117
US	2001-764900	B1 20010117
US	2001-764902	A2 20010117
US	2001-764903	A1 20010117
US	2001-764904	A1 20010117
US	2001-764905	A2 20010117
WO	2001-US1239	A2 20010117
WO	2001-US1307	A2 20010117
WO	2001-US1312	A2 20010117
WO	2001-US1320	A2 20010117
WO	2001-US1329	A2 20010117
WO	2001-US1334	A2 20010117
WO	2001-US1336	A2 20010117
WO	2001-US1339	A2 20010117
WO	2001-US1340	A2 20010117
WO	2001-US1341	W 20010117
WO	2001-US1344	A2 20010117
WO	2001-US1345	A2 20010117
WO	2001-US1347	A2 20010117
WO	2001-US1348	A2 20010117
WO	2001-US1360	A2 20010117

AB The present invention relates to novel proteins. More specifically, 461 isolated nucleic acid mols. are provided encoding novel polypeptides. Novel polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and **recombinant** and synthetic methods for producing human polynucleotides and/or polypeptides, and antibodies. The invention further relates to **diagnostic** and therapeutic methods useful for **diagnosing**, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to **screening** methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compns. for inhibiting or enhancing the production and function of the polypeptides of the present invention.

IT **353326-61-1P**, Protein (human clone HTGAW31 fragment)
353513-54-9P, Protein (human clone fragment)
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); USES (Uses)
(amino acid sequence; human nucleic acids and their encoded proteins and antibodies)

L4 ANSWER 14 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 03 Aug 2001

ACCESSION NUMBER: 2001:564770 HCAPLUS
DOCUMENT NUMBER: 135:163373
TITLE: Protein and cDNA sequences of potential novel human transport proteins
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: PCT Int. Appl., 811 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 93
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001054472	A2	20010802	WO 2001-US1307	20010117
WO 2001054472	A3	20020131		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, NE, SN, TD, TG				
AU 2001062899	A5	20011018	AU 2001-62899	20010117
US 2002042096	A1	20020411	US 2001-764887	20010117
US 2002077270	A1	20020620	US 2001-764848	20010117
US 2002086811	A1	20020704	US 2001-764861	20010117
US 2003171252	A9	20030911		
US 2002086820	A1	20020704	US 2001-764862	20010117
US 2003092611	A9	20030515		
US 2002086821	A1	20020704	US 2001-764881	20010117
US 2003125246	A9	20030703		
US 2002086822	A1	20020704	US 2001-764886	20010117
US 2003139327	A9	20030724		
US 2002086823	A1	20020704	US 2001-764889	20010117
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US 2002090615	A1	20020711	US 2001-764878	20010117
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US 2002147140	A1	20021010	US 2001-764877	20010117
US 2002151479	A1	20021017	US 2001-764873	20010117
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EP 1254272	A2	20021106	EP 2001-937134	20010117
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2002164685	A1	20021107	US 2001-764857	20010117
US 2002173454	A1	20021121	US 2001-764904	20010117
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US 2003050231	A1	20030313	US 2001-764872	20010117
AU 2001041411	A5	20010820	AU 2001-41411	20010208
US 2002045230	A1	20020418	US 2001-908711	20010720

US 2003013649	A1	20030116	US 2001-989442		20011121
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US 2003054373	A1	20030320	US 2002-91572		20020307
US 2003054375	A1	20030320	US 2002-92154		20020307
US 2003059908	A1	20030327	US 2002-91504		20020307
US 2003068627	A1	20030410	US 2002-91458		20020307
US 2003077606	A1	20030424	US 2002-91438		20020307
US 2003082681	A1	20030501	US 2002-91391		20020307
US 2003054377	A1	20030320	US 2002-102627		20020322
US 2003082758	A1	20030501	US 2002-103313		20020322
US 2003054379	A1	20030320	US 2002-116016		20020405
US 2003092615	A1	20030515	US 2002-115928		20020405
US 2003059875	A1	20030327	US 2002-125540		20020419
PRIORITY APPLN. INFO.:			US 2000-179065P	P	20000131
			US 2000-180628P	P	20000204
			US 2000-184664P	P	20000224
			US 2000-186350P	P	20000302
			US 2000-189874P	P	20000316
			US 2000-190076P	P	20000317
			US 2000-198123P	P	20000418
			US 2000-205515P	P	20000519
			US 2000-209467P	P	20000607
			US 2000-214886P	P	20000628
			US 2000-215135P	P	20000630
			US 2000-216647P	P	20000707
			US 2000-216880P	P	20000707
			US 2000-217487P	P	20000711
			US 2000-217496P	P	20000711
			US 2000-218290P	P	20000714
			US 2000-220963P	P	20000726
			US 2000-220964P	P	20000726
			US 2000-225270P	P	20000814
			US 2000-225757P	P	20000814
			US 2000-224518P	P	20000814
			US 2000-224519P	P	20000814
			US 2000-225267P	P	20000814
			US 2000-225268P	P	20000814
			US 2000-225447P	P	20000814
			US 2000-225758P	P	20000814
			US 2000-226868P	P	20000822
			US 2000-228924P	P	20000830
			US 2000-229287P	P	20000901
			US 2000-229343P	P	20000901

Searcher : Shears 571-272-2528

US 2000-229344P	P	20000901
US 2000-229345P	P	20000901
US 2000-229509P	P	20000905
US 2000-229513P	P	20000905
US 2000-231413P	P	20000908
US 2000-232398P	P	20000914
US 2000-234223P	P	20000921
US 2000-234274P	P	20000921
US 2000-234997P	P	20000925
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US 2000-236327P	P	20000929
US 2000-236367P	P	20000929
US 2000-236368P	P	20000929
US 2000-236369P	P	20000929
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US 2000-237037P	P	20001002
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US 2000-237040P	P	20001002
US 2000-241221P	P	20001020
US 2000-241785P	P	20001020
US 2000-241786P	P	20001020
US 2000-241809P	P	20001020
US 2000-244617P	P	20001101
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US 2000-246523P	P	20001108
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US 2000-249207P	P	20001117
US 2000-249208P	P	20001117
US 2000-249210P	P	20001117
US 2000-249211P	P	20001117
US 2000-249212P	P	20001117
US 2000-249213P	P	20001117
US 2000-249215P	P	20001117
US 2000-249216P	P	20001117
US 2000-249217P	P	20001117
US 2000-249218P	P	20001117
US 2000-249244P	P	20001117
US 2000-249245P	P	20001117
US 2000-249297P	P	20001117
US 2000-249299P	P	20001117
US 2000-249300P	P	20001117
US 2000-250160P	P	20001201
US 2000-251856P	P	20001208
US 2000-251868P	P	20001208
US 2000-251869P	P	20001208
US 2000-251990P	P	20001208
US 2001-764847	B1	20010117
US 2001-764848	B1	20010117
US 2001-764850	B1	20010117
US 2001-764852	B1	20010117
US 2001-764853	A2	20010117
US 2001-764854	B1	20010117
US 2001-764855	B1	20010117

Searcher : Shears 571-272-2528

US	2001-764856	A2 20010117
US	2001-764857	B1 20010117
US	2001-764860	B1 20010117
US	2001-764861	A1 20010117
US	2001-764862	A1 20010117
US	2001-764863	B1 20010117
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US	2001-764870	A2 20010117
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US	2001-764874	A2 20010117
US	2001-764878	A1 20010117
US	2001-764879	B1 20010117
US	2001-764882	A2 20010117
US	2001-764885	B1 20010117
US	2001-764887	B1 20010117
US	2001-764888	A2 20010117
US	2001-764889	A1 20010117
US	2001-764891	A2 20010117
US	2001-764892	A2 20010117
US	2001-764893	B1 20010117
US	2001-764896	A2 20010117
US	2001-764898	A2 20010117
US	2001-764900	B1 20010117
US	2001-764902	A2 20010117
US	2001-764903	A1 20010117
US	2001-764904	A1 20010117
US	2001-764905	A2 20010117
WO	2001-US1239	A2 20010117
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WO	2001-US1312	A2 20010117
WO	2001-US1320	A2 20010117
WO	2001-US1329	A2 20010117
WO	2001-US1334	A2 20010117
WO	2001-US1336	A2 20010117
WO	2001-US1339	A2 20010117
WO	2001-US1340	A2 20010117
WO	2001-US1341	A2 20010117
WO	2001-US1344	A2 20010117
WO	2001-US1345	A2 20010117
WO	2001-US1347	A2 20010117
WO	2001-US1348	A2 20010117
WO	2001-US1360	A2 20010117

AB The present invention relates to novel polynucleotides and the polypeptides encoded by these polynucleotides, and the use of such proteins, which are potential transport proteins, for **diagnosing**, treating, preventing disorders related to these novel peptides. More specifically, 284 isolated cDNA mols. are provided encoding novel polypeptides. Novel polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and **recombinant** and synthetic methods for producing novel human polynucleotides and/or polypeptides. The invention further relates to **screening**

methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compns. for inhibiting the production and function of the polypeptides of the present invention.

IT **353550-30-8 353553-06-7**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; protein and cDNA sequences of potential novel human transport proteins)

L4 ANSWER 15 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 06 Apr 2001
ACCESSION NUMBER: 2001:247142 HCAPLUS
DOCUMENT NUMBER: 134:306971
TITLE: Colon and colon cancer associated cDNAs and proteins and their use in diagnosis and treatment of colon cancer
INVENTOR(S): Ruben, Steven M.; Barash, Steven C.; Birse, Charles E.; Rosen, Craig A.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: PCT Int. Appl., 9787 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001022920	A2	20010405	WO 2000-US26524	20000928
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2000077215	A5	20010430	AU 2000-77215	20000928
EP 1265582	A2	20021218	EP 2000-966944	20000928
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
US 2003109690	A1	20030612	US 2002-106698	20020327
US 2003109690	A1	20030612	US 2002-106698	20020327

PRIORITY APPLN. INFO.:
US 1999-157137P P 19990929
US 1999-163280P P 19991103
WO 2000-US26524 W 20000928
US 2002-106698 A 20020327

AB This invention relates to newly identified colon or colon cancer related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "colon cancer antigens", and the use of such colon cancer antigens for targeting specific cell types and/or diagnosing, detecting, preventing and treating disorders of the colon, particularly the presence of colon

cancer and colon cancer metastases. This invention relates to colon cancer antigens as well as vectors, host cells, antibodies directed to colon cancer antigens and the recombinant or synthetic methods for producing the same. Also provided are diagnostic methods for diagnosing and treating, preventing and/or prognosing disorders related to the colon, including colon cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of colon cancer antigens of the invention. The present invention further relates to inhibiting the production and function of the polypeptides of the present invention.

L4 ANSWER 16 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 22 Sep 2000
ACCESSION NUMBER: 2000:666903 HCAPLUS
DOCUMENT NUMBER: 133:233618
TITLE: Human cancer-associated gene sequences and polypeptides
INVENTOR(S): Rosen, Craig A.; Ruben, Steven M.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: PCT Int. Appl., 2352 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 10
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000055350	A1	20000921	WO 2000-US5882	20000308
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1163358	A1	20011219	EP 2000-917770	20000308
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2004508001	T2	20040318	JP 2000-605767	20000308
US 2002052308	A1	20020502	US 2001-925301	20010810

PRIORITY APPLN. INFO.: US 1999-124270P P 19990312
WO 2000-US5882 W 20000308

AB This invention relates to 842 newly identified cancer-related cDNAs and the polypeptides encoded by these polynucleotides herein collectively known as "cancer antigens", and to the complete gene sequences associated therewith and to the expression products thereof, as well as the use of such cancer antigens for **detection**, prevention and treatment of disorders of tissue-specific disorders, particularly the presence of cancer. This invention relates to the cancer antigens as well as vectors, host cells, antibodies directed to cancer antigens, and **recombinant** and synthetic methods for producing the same. Also provided are **diagnostic**

methods for **diagnosing** and treating, preventing and/or prognosing tissue-specific disorders, including cancer, and therapeutic methods for treating such disorders. The invention further relates to **screening** methods for identifying agonists and antagonists of cancer antigens of the invention. The present invention further relates to methods and/or compns. for inhibiting the production and/or function of the polypeptides of the present invention.

IT **292882-95-2**
RL: ANT (Analyte); BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(amino acid sequence; human cancer-associated gene sequences and polypeptides)

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 17 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 19 Feb 1999
ACCESSION NUMBER: 1999:112693 HCAPLUS
DOCUMENT NUMBER: 130:308093
TITLE: Rat NAP1: cDNA cloning and upregulation by Mpl ligand
AUTHOR(S): Cataldo, Leah M.; Zhang, Ying; Lu, Jun; Ravid, Katya
CORPORATE SOURCE: Department of Biochemistry, Whitaker Cardiovascular Institute, Boston University School of Medicine, Boston, MA, 02118, USA
SOURCE: Gene (1999), 226(2), 355-364
CODEN: GENED6; ISSN: 0378-1119
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The Mpl ligand is a hematopoietic cytokine which exerts its effects through association with the c-Mpl receptor. It regulates the proliferation, polyploidization and maturation of platelet precursors, the megakaryocytes. Using a differential display polymerase chain reaction (PCR) approach, we have identified an mRNA, belonging to a family of nucleosome assembly proteins, whose expression is upregulated in response to Mpl ligand. Multiple size classes of this mRNA (1.7, 2.5 and 4.3 kb) are readily **detected** in rat primary bone marrow cells and hematopoietic tissues. The size classes are also expressed to different extents in cell lines of all hematopoietic lineages. We isolated the full-length cDNA encoding the rat megakaryocyte 1.7 kb mRNA, referred to as rNAP1. Bacterially expressed **recombinant** protein encoded by the 1.7 kb cDNA facilitates the formation of nucleosomes on relaxed circular DNA in vitro. Our data indicate that rNAPs, which may facilitate chromatin reorganization, are upregulated by Mpl ligand. It is possible that NAPs contribute to Mpl ligand's induced effects on hematopoietic cells.

IT **223503-67-1**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological

study)
(amino acid sequence; cDNA cloning and upregulation by Mpl ligand of rat NAP1 (nucleosome assembly protein 1))
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 18 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 30 May 1998
ACCESSION NUMBER: 1998:323143 HCAPLUS
DOCUMENT NUMBER: 129:40139
TITLE: Novel polynucleotide and polypeptide sequences of Streptococcal pneumoniae for preparing antibody and **screening** antimicrobial compounds
INVENTOR(S): Black, Michael Terance; Hodgson, John Edward; Knowles, David Justin Charles; Lonetto, Michael Arthur; Nicholas, Richard Oakley; Reid, Robert H., Jr.; Zarfos, Phillip N.
PATENT ASSIGNEE(S): Smithkline Beecham Corp., USA; Smithkline Beecham PLC; Black, Michael Terance; Hodgson, John Edward; Knowles, David Justin Charles; Lonetto, Michael Arthur; Nicholas, Richard Oakley; Reid, Robert H., Jr.; Zarfos, Phillip N.
SOURCE: PCT Int. Appl., 130 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9819689	A1	19980514	WO 1997-US19226	19971027
W: CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1007069	A1	20000614	EP 1997-911905	19971027
R: BE, CH, DE, DK, FR, GB, IT, LI, NL				
JP 2001510989	T2	20010807	JP 1998-521476	19971027
PRIORITY APPLN. INFO.:			US 1996-29930P P	19961101
			WO 1997-US19226 W	19971027

AB This invention relates to newly identified Streptococcal polynucleotides, polypeptides encoded by such polynucleotides, the uses of such polynucleotides and polypeptides, as well as the production of such polynucleotides and polypeptides and **recombinant** host cells transformed with the polynucleotides. This invention also relates to inhibiting the biosynthesis or action of such polynucleotides or polypeptides and to the use of such inhibitors in therapy.
IT **208408-72-4**
RL: PRP (Properties)
(amino acid sequence; novel polynucleotide and polypeptide sequences of Streptococcal pneumoniae for preparing antibody and **screening** antimicrobial compds.)
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR

THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 19 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 19 Mar 1997
ACCESSION NUMBER: 1997:181090 HCAPLUS
DOCUMENT NUMBER: 126:176879
TITLE: Nucleic acid and amino acid sequences relating to Helicobacter pylori for **diagnostics** and therapeutics
INVENTOR(S): Smith, Douglas; Berglindh, O. Thomas; Mellgaerd, Bjoern L.
PATENT ASSIGNEE(S): Astra Aktiebolag, Swed.
SOURCE: PCT Int. Appl., 1479 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9640893	A1	19961219	WO 1996-US9122	19960606
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI				
CA 2223395	AA	19961219	CA 1996-2223395	19960606
AU 9663278	A1	19961230	AU 1996-63278	19960606
AU 710880	B2	19990930		
EP 842270	A1	19980520	EP 1996-922393	19960606
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI				
CN 1186516	A	19980701	CN 1996-195715	19960606
JP 11504220	T2	19990420	JP 1997-501519	19960606
BR 9609430	A	19990824	BR 1996-9430	19960606
ZA 9604869	A	19970707	ZA 1996-4869	19960607
NO 9705745	A	19980209	NO 1997-5745	19971205
AU 9937956	A1	19990826	AU 1999-37956	19990705
AU 713692	B2	19991209		
AU 9937960	A1	19990826	AU 1999-37960	19990705

PRIORITY APPLN. INFO.:		
US 1995-487032	A	19950607
US 1995-561469	A	19951117
US 1996-630405	A	19960401
AU 1996-63278	A3	19960606
WO 1996-US9122	W	19960606

AB **Recombinant** or substantially pure prepns. of H. pylori polypeptides and the nucleic acids encoding the polypeptides are described. Thus, H. pylori genomic DNA was mech. sheared by nebulization to a median size of 2 kb, fractionated by gel electrophoresis, blunt-ended and ligated to adapter oligonucleotides, and cloned into each of 20 different pMPX vectors to construct a series of shotgun subclone libraries. DNA sequencing was achieved using multiplex sequencing procedures. The H. pylori

sequences were analyzed for the presence of open reading frames comprising at least 180 nucleotides; as a result of the anal. of ORFs based on stop-to-stop codon reads, predicted coding regions were defined by evaluating the coding potential of such sequences with the program GENEMARK. About 452 sep. stop-to-stop codon ORFs are presented, and 507 predicted coding regions. The pET System was used for cloning and expression of **recombinant** proteins in Escherichia coli. Several of the specific polypeptides were thus expressed in transformed bacteria, purified, shown to generate antibodies, and analyzed as vaccine candidates. The H. pylori nucleic acids and polypeptides are useful for **diagnostics** and vaccine compns. An exptl. knock-out protocol is presented for **determination** of essential H. pylori genes as potential therapeutic targets.

IT **186984-83-8P**
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; nucleic acid and amino acid sequences relating to Helicobacter pylori for **diagnostics** and therapeutics)

E1 THROUGH E22 ASSIGNED

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L6 22 L1 AND L5

L6 ANSWER 1 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **674875-34-4** REGISTRY
CN INDEX NAME NOT YET ASSIGNED
CI MAN
SQL 392

SEQ
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  1 MADIDNKEQT ELDQQDMEDV EDVEEEETGE DANSKARQLT AQMMQNPQVL
 51 AALQERLDDL VGTPTGYIES LPKVVKRRVN ALKNLQVKCA QIEAKFYEEV
101 HELERKYAAL YQPLFDKRSD IINATYEPTE EECEWKVEEE DISGDLKEKA
                                                =====
151 KLEEEKKDEE KEDPKGIPEF WLTVFKNVDL LNDMLQEHDE PILKHLKDIK
    =
201 VKFSDAGQPM SFTLEFYFEP NEFFTNEVLT KTYKMRSEPD ESDPFSFDGP
251 EIMGCTGCLI DWKKGKNVTL KTIKKKQKHK GRGTVRTVTK TVPNDSFFNF
301 FTPPEVPENG ELDDDAEAIL TADFEIGHFL RERIIPRSVL YFTGEAIEDD
351 DDDYDEQGEE ADDEFESREA DEDNDPDYEP KKGQNPAECK QQ
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HITS AT: 146-151

REFERENCE 1: 140:284548

L6 ANSWER 2 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **660003-26-9** REGISTRY
CN Protein (human gene 32229) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 58: PN: US20040033509 SEQID: 121 claimed protein
CI MAN
SQL 797

SEQ 1 MEIPKDSLQK YLKDLLGIQT TGPLELLQFD HGQSNPTYYI RLANRDLVLR
51 KKPPGTLLPS AHAIEREFRI MKALANAGVP VPNVLDLCED SSVIGTPFYV
101 MEYCPGLIYK DPSLPGLEPS HRRAIYTAMN TVLCKIHSVD LQAVGLEDYG
151 KQGDYIPRQV RTWVKQYRAS ETSTIPAMER LIEWLPLHLP RQQRTTVVHG
201 DFRLDNLVFH PEEPEVLAVL DWELSTLGDP LADVAYSCLA HYLPSSFPVL
251 RGINDCDLTQ LGIPAAEEYF RMYCLQMGLP PTENWNFYMA FSFFRVAAIL
301 QGVYKRSLTG QASSTYAEQT GKLTEFVSNL AWDFAVKEGF RVFKEMPFTN
351 PLTRSYHTWA RPQSQWCPTG SRSYSSVPEA SPAHTSRGGL VISPESLSPP
401 VRELYHRLKH FMEQRVYPAE PELQSHQASA ARWSPSPLIE DLKEKAKAEG
====== 451 LWNLFLPLEA DPEKKYGAGL TNVEYAHLCE LMGTSLYAPE VCNCSAPDTG
501 NMELLVRYGT EAQKARWLIP LLEGKARSCF AMTEPQVASS DATNIEASIR
551 EEDSFYVING HKWWITGILD PRCQLCVFMG KTDPHAPRHR QQSVLLVPMD
601 TPGIKIIRPL TVYGLEDAPG GHGEVRFEHV RVPKENMVLG PGRGFEIAQG
651 RLGPGRIHHC MRLIGFSERA LALMKARVKS RLAFGKPLVE QGTVLADIAQ
701 SRVEIEQARL LVLRAAHLMD LAGNKAAALD IAMIKMVAPS MASRVIDRAI
751 QAFGAAGLSS DYPLAQFFTW ARALRFADGP DEVHRATVAK LELKHRI
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RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 140:176312

L6 ANSWER 3 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **642131-60-0** REGISTRY
CN Protein (Moraxella catarrhalis strain 98-4362 clone US6673910-SEQID-2189 N-terminal fragment) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2189: PN: US6673910 SEQID: 2189 claimed protein
CI MAN
SQL 171

SEQ 1 YKSVDQEAMD ALMKIEEFRS LNDDIEVVIN RSEKIKGLKE KAKDNDGELS
=== === 51 KEDKKTLSDE EKEQKSLRKQ IQEKLIKFAT RIPIFMYLSD YREHSLKDVI
101 TVLEPELFTR VTGLTQADFS LLVSLNVFDE AVMNDAVYKF KRYEDASLEY
151 AGIDKKEGYV GLYNTQIKRQ W
HITS AT: 38-43

REFERENCE 1: 140:88747

L6 ANSWER 4 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **604828-86-6** REGISTRY
CN Protein (Candida albicans gene CaYKL099C) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1971: PN: US20030180953 SEQID: 7107 claimed protein
CI MAN

```
SQL  247

SEQ      1 MAKLVHNVQK KQHRERSQTQ SRARYGLLEK KKDYKLRAAD YHKKQAALKA
        51 LKEKAKLHNP DEYYHAMTRK KTDDKGILIS ERDNEVLSVE QAKLLKTQDV
           ======
       101 NYIRTMRLNE LKKIEKEKEG KLFGASGKHT VFVDSIEEQE SFNPEEFFDT
       151 DAALLDNREN RLRMNQLYDN SGLLTSNDLD IDTKNKLDLK KLKQYKLLQR
       201 RLKKEKELKE VESIMSKNLE KMKKGNKKKV VDSNGKVHFK WKNERKR
HITS AT:   51-56

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  139:272071

L6   ANSWER 5 OF 22  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   536052-37-6  REGISTRY
CN   Colon tumor-associated protein (human clone US20030109690-SEQID-4620
     fragment) (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   333: PN: US20030109690 SEQID: 4620 claimed protein
CI   MAN
SQL  406

SEQ      1 VTACAAPAAW LPILVADIWS SYNMADIDNK EQSELDQDLD DVEEVEEEET
        51 GEETKLKARQ LTVQMMQNPQ ILAALQERLD GLVETPTGYI ESLPRVVKRR
       101 VNALKNLQVK CAQIEAKFYE EVHDLERKYA VLYQPLFDKR FEIINAIYEP
       151 TEEECEWKPD EEDEISEELK EKAKIEDEKK DEEKEDPKGI PEFWLTVFKN
                              == ====
       201 VDLLSDMVQE HDEPILKHLK DIKVKFSDAG QPMSFVLEFH FEPNEYFTNE
       251 VLTKTYRMRS EPDDSDPFSF DGPEIMGCTG CQIDWKKGKN VTLKTIKKKQ
       301 KHKGRGTVRT VTKTVSNDSF FNFFAPPEVP ESGDLDDDAE AILAADFEIG
       351 HFLRERIIPR SVLYFTGEAI EDDDDDYDEE GEEADEGYQL FEEVKSCSKL
       401 FQRWLQ
HITS AT:   169-174

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  139:2147

L6   ANSWER 6 OF 22  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   443161-46-4  REGISTRY
CN   Protein (Candida albicans clone Sa386383_7107 gene fragment) (9CI)
     (CA INDEX NAME)
OTHER NAMES:
CN   107: PN: WO02053728 SEQID: 7107 claimed protein
CI   MAN
SQL  247

SEQ      1 MAKLVHNVQK KQHRERSQTQ SRARYGLLEK KKDYKLRAAD YHKKQAALKA
        51 LKEKAKLHNP DEYYHAMTRK KTDDKGILIS ERDNEVLSVE QAKLLKTQDV
           ======
       101 NYIRTMRLNE LKKIEKEKEG KLFGASGKHT VFVDSIEEQE SFNPEEFFDT
       151 DAALLDNREN RLRMNQLYDN SGLLTSNDLD IDTKNKLDLK KLKQYKLLQR
       201 RLKKEKELKE VESIMSKNLE KMKKGNKKKV VDSNGKVHFK WKNERKR
HITS AT:   51-56
```

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:104826

L6 ANSWER 7 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **420277-73-2** REGISTRY
CN Dehydrogenase (human clone Fbh21686 isoenzyme 4 precursor) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 11: PN: US20020052032 SEQID: 11 claimed protein
CI MAN
SQL 322

```
SEQ      1 MSLRPRRACA QLLWHPAAGM ASWAKGRSYL APGLLQGQVA IVTGGATGIG
        51 KAIVKELLEL GSNVVIASRK LERLKSAADE LQANLPPTKQ ARVIPIQCNI
       101 RNEEEVNNLV KSTLDTFGKI NFLVNNGGGQ FLSPAEHISS KGWHAVLETN
       151 LTGTFYMCKA VYSSWMKEHG GSIVNIIVPT KAGFPLAVHS GAARAGVYNL
       201 TKSLALEWAC SGIRINCVAP GVIYSQTAVE NYGSWGQSFF EGSFQKIPAK
       251 RIGVPEEVSS VVCFLLSPAA SFITGQSVDV DGGRSLYTHS YEVPDHDNWP
       301 KGAGDLSVVK KMKETLKEKA KL
                           ===== =
HITS AT:   316-321
```

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 136:352023

L6 ANSWER 8 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **416229-10-2** REGISTRY
CN Protein (human clone 32229 acyl coenzyme A dehydrogenase sequence homolog) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2: PN: WO0233061 SEQID: 2 claimed protein
CI MAN
SQL 797

```
SEQ      1 MEIPKDSLQK YLKDLLGIQT TGPLELLQFD HGQSNPTYYI RLANRDLVLR
        51 KKPPGTLLPS AHAIEREFRI MKALANAGVP VPNVLDLCED SSVIGTPFYV
       101 MEYCPGLIYK DPSLPGLEPS HRRAIYTAMN TVLCKIHSVD LQAVGLEDYG
       151 KQGDYIPRQV RTWVKQYRAS ETSTIPAMER LIEWLPLHLP RQQRTTVVHG
       201 DFRLDNLVFH PEEPEVLAVL DWELSTLGDP LADVAYSCLA HYLPSSFPVL
       251 RGINDCDLTQ LGIPAAEEYF RMYCLQMGLP PTENWNFYMA FSFFRVAAIL
       301 QGVYKRSLTG QASSTYAEQT GKLTEFVSNL AWDFAVKEGF RVFKEMPFTN
       351 PLTRSYHTWA RPQSQWCPTG SRSYSSVPEA SPAHTSRGGL VISPESLSPP
       401 VRELYHRLKH FMEQRVYPAE PELQSHQASA ARWSPSPLIE DLKEKAKAEG
                                                    ======
       451 LWNLFLPLEA DPEKKYGAGL TNVEYAHLCE LMGTSLYAPE VCNCSAPDTG
       501 NMELLVRYGT EAQKARWLIP LLEGKARSCF AMTEPQVASS DATNIEASIR
       551 EEDSFYVING HKWWITGILD PRCQLCVFMG KTDPHAPRHR QQSVLLVPMD
       601 TPGIKIIRPL TVYGLEDAPG GHGEVRFEHV RVPKENMVLG PGRGFEIAQG
       651 RLGPGRIHHC MRLIGFSERA LALMKARVKS RLAFGKPLVE QGTVLADIAQ
       701 SRVEIEQARL LVLRAAHLMD LAGNKAAALD IAMIKMVAPS MASRVIDRAI
       751 QAFGAAGLSS DYPLAQFFTW ARALRFADGP DEVHRATVAK LELKHRI
HITS AT:   442-447
```

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 136:336297

L6 ANSWER 9 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **410117-83-8** REGISTRY
CN 2: PN: US20020042371 FIGURE: 19 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 303

```
SEQ      1 MASWAKGRSY LAPGLLQGQV AIVTGGATGI GKAIVKELLE LGSNVVIASR
        51 KLERLKSAAD ELQANLPPTK QARVIPIQCN IRNEEEVNNL VKSTLDTFGK
       101 INFLVNNGGG QFLSPAEHIS SKGWHAVLET NLTGTFYMCK AVYSSWMKEH
       151 GGSIVNIIVP TKAGFPLAVH SGAARAGVYN LTKSLALEWA CSGIRINCVA
       201 PGVIYSQTAV ENYGSWGQSF FEGSFQKIPA KRIGVPEEVS SVVCFLLSPA
       251 ASFITGQSVD VDGGRSLYTH SYEVPDHDNW PKGAGDLSVV KKMKETLKEK
                                                              ====
       301 AKL
           ==
```

HITS AT: 297-302

REFERENCE 1: 136:305208

L6 ANSWER 10 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **410115-99-0** REGISTRY
CN dehydrogenase (human clone 21686 sequence homolog) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 13: PN: US20020042371 SEQID: 11 claimed protein
CI MAN
SQL 322

```
SEQ      1 MSLRPRRACA QLLWHPAAGM ASWAKGRSYL APGLLQGQVA IVTGGATGIG
        51 KAIVKELLEL GSNVVIASRK LERLKSAADE LQANLPPTKQ ARVIPIQCNI
       101 RNEEEVNNLV KSTLDTFGKI NFLVNNGGGQ FLSPAEHISS KGWHAVLETN
       151 LTGTFYMCKA VYSSWMKEHG GSIVNIIVPT KAGFPLAVHS GAARAGVYNL
       201 TKSLALEWAC SGIRINCVAP GVIYSQTAVE NYGSWGQSFF EGSFQKIPAK
       251 RIGVPEEVSS VVCFLLSPAA SFITGQSVDV DGGRSLYTHS YEVPDHDNWP
       301 KGAGDLSVVK KMKETLKEKA KL
                   ===== =
```

HITS AT: 316-321

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 136:305208

L6 ANSWER 11 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **382665-85-2** REGISTRY
CN Protein (human clone XD4) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 17: PN: WO0196523 SEQID: 17 claimed protein
CN Protein (human clone XD4 RNA-binding protein sequence homolog)
CI MAN
SQL 174

```
SEQ      1 MADVLDLHEA GGEDFAMDED GDESIHKLKE KAKKRKGRGF GSEEGSRARM
```

```
                                          === ===
       51 REDYDSVEQD GDEPGPQRSV EGWILFVTGV HEEATEEDIH DKFAEYGEIK
      101 NIHLNLDRRT GYLKGYTLVE YETYKEAQAA MEGLNGQDLM GQPISVDWCF
      151 VRGPPKGKRR GGRRRSRSPD RRRR
HITS AT:    28-33

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  136:52364

L6   ANSWER 12 OF 22  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   379739-09-0  REGISTRY
CN   Anthocyanin 3'-O-glucosyltransferase (Gentiana triflora clone
     pGe'GT7) (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   2: PN: WO0192509 SEQID: 2 claimed protein
CI   MAN
SQL  482

SEQ      1 MDQLHVFFFP FLANGHILPT IDMAKLFSSR GVKATLITTH NNSAIFLKAI
        51 NRSKILGFDI SVLTIKFPSA EFGLPEGYET ADQARSIDMM DEFFRACILL
       101 QEPLEELLKE HRPQALVADL FFYWANDAAA KFGIPRLLFH GSSSFAMIAA
       151 ESVRRNKPYK NLSSDSDPFV VPDIPDKIIL TKSQVPTPDE TEENNTHITE
       201 MWKNISESEN DCYGVIVNSF YELEPDYVDY CKNVLGRRAW HIGPLSLCNN
       251 EGEDVAERGK KSDIDAHECL NWLDSKNPDS VVYVCFGSMA NFNAAQLHEL
       301 AMGLEESGQE FIWVVRTCVD EEDESKWFPD GFEKRVQENN KGLIIKGWAP
       351 QVLILEHEAV GAFVSHCGWN STLEGICGGV AMVTWPLFAE QFYNEKLMTD
       401 ILRTGVSVGS LQWSRVTTSA VVVKRESISK AVRRLMAEEE GVDIRNRAKA
       451 LKEKAKKAVE GGGSSYSDLS ALLVELSSYP HN
           ======
HITS AT:    451-456

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  136:33913

L6   ANSWER 13 OF 22  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   364639-16-7  REGISTRY
CN   15: PN: WO0172976 FIGURE: 19 unclaimed sequence (9CI)  (CA INDEX
     NAME)
CI   MAN
SQL  303

SEQ      1 MASWAKGRSY LAPGLLQGQV AIVTGGATGI GKAIVKELLE LGSNVVIASR
        51 KLERLKSAAD ELQANLPPTK QARVIPIQCN IRNEEEVNNL VKSTLDTFGK
       101 INFLVNNGGG QFLSPAEHIS SKGWHAVLET NLTGTGYMCK AVYSSWMKEH
       151 GGSIVNIIVP TKAGFPLAVH SGAARAGVYN LTKSLALENA CSGIRINCVA
       201 PGVIYSQTAV ENYGSWGQSF FEGSFQKIPA KRIGVPEEVS SVVCFLLSPA
       251 ASFITGQSVD VDGGRSLYTH SYEVPDHDNW PKGAGDLSVV KKMKETLKEK
                                                           ====
       301 AKL
           ==
HITS AT:    297-302

REFERENCE   1:  135:285001
```

L6 ANSWER 14 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **364638-73-3** REGISTRY
CN Protein (human clone 21686 dehydrogenase sequence homolog) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 13: PN: WO0172976 FIGURE: 16 claimed sequence
CI MAN
SQL 322

SEQ 1 MSLRPRRACA QLLWHPAAGM ASWAKGRSYL APGLLQGQVA IVTGGATGIG
51 KAIVKELLEL GSNVVIASRK LERLKSAADE LQANLPPTKQ ARVIPIQCNI
101 RNEEEVNNLV KSTLDTFGKI NFLVNNGGGQ FLSPAEHISS KGWHAVLETN
151 LTGTFYMCKA VYSSWMKEHG GSIVNIIVPT KAGFPLAVHS GAARAGVYNL
201 TKSLALEWAC SGIRINCVAP GVIYSQTAVE NYGSWGQSFF EGSFQKIPAK
251 RIGVPEEVSS VVCFLLSPAA SFITGQSVDV DGGRSLYTHS YEVPDHDNWP
301 KGAGDLSVVK KMKETLKEKA KL
===== =

HITS AT: 316-321

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:285001

L6 ANSWER 15 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **353553-06-7** REGISTRY
CN Protein (human clone HKAAK01 189-amino acid) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 824: PN: WO0154472 SEQID: 833 claimed protein
CI MAN
SQL 189

SEQ 1 QYDRFRSPRA RIEGEMADVL DLHEAGGEDF AMDEDGDESI HKLKEKAKKR
======
51 KGRGFGSEEG SRARMREDYD SVEQDGDEPG PQRSVEGWIL FVTGVHEEAT
101 EEDIHDKFAE YGEIKNIHLN LDRRTGYLKG YTLVEYETYK EAQAAMEGLN
151 GQDLMGQPIS VDWCFVRGPP KGKRRGGRRR SRSPDRRRR
HITS AT: 43-48

REFERENCE 1: 135:163373

L6 ANSWER 16 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **353550-30-8** REGISTRY
CN Protein (human clone HKAAK01 184-amino acid) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 565: PN: WO0154472 SEQID: 574 claimed protein
CI MAN
SQL 184

SEQ 1 VDPRVRIEGE MADVLDLHEA GGEDFAMDED GDESIHKLKE KAKKRKGRGF
=== ===
51 GSEEGSRARM REDYDSVEQD GDEPGPQRSV EGWILFVTGV HEEATEEDIH
101 DKFAEYGEIK NIHLNLDRRT GYLKGYTLVE YETYKEAQAA MEGLNGQDLM
151 GQPISVDWCF VRGPPKGKRR GGRRRSRSPD RRRR
HITS AT: 38-43

REFERENCE 1: 135:163373

L6 ANSWER 17 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **353513-54-9** REGISTRY
CN Protein (human clone fragment) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1547: PN: WO0155322 SEQID: 1549 claimed protein
CI MAN
SQL 60

SEQ 1 SSSPRRIANL VQKRYELLEH KKRWASMSEE QRXEYLKKKR EELKKKLKEK
====
51 AKERRERERE
==
HITS AT: 47-52

REFERENCE 1: 135:163399

L6 ANSWER 18 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **353326-61-1** REGISTRY
CN Protein (human clone HTGAW31 fragment) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1130: PN: WO0155322 SEQID: 1134 claimed protein
CI MAN
SQL 141

SEQ 1 SSKPHKHLPP AALHLIAYYK ENKDREDKRS ALSCVISKTA RLLSSEDRAR
51 LPEELRSLVQ KRYELLEHKK RWASMSEEQR KEYLKKKREE LKKKLKEKAK
======
101 ERREXEMLER LEKQKRYEDQ ELTGKNLPAF RLVDTPEGLP N
HITS AT: 95-100

REFERENCE 1: 135:163399

L6 ANSWER 19 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **292882-95-2** REGISTRY
CN Tumor-associated protein (human clone HDTLJ39) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1200: PN: WO0122920 SEQID: 4610 claimed protein
CN 162: PN: WO0055350 SEQID: 1320 claimed protein
CN Protein (human clone HE2CH58 colon cancer-associated)
CI MAN
SQL 406

SEQ 1 VTACAAPAAW LPILVADIWS SYNMADIDNK EQSELDQDLD DVEEVEEEET
51 GEETKLKARQ LTVQMMQNPQ ILAALQERLD GLVETPTGYI ESLPRVVKRR
101 VNALKNLQVK CAQIEAKFYE EVHDLERKYA VLYQPLFDKR FEIINAIYEP
151 TEEECEWKPD EEDEISEELK EKAKIEDEKK DEEKEDPKGI PEFWLTVFKN
== ====
201 VDLLSDMVQE HDEPILKHLK DIKVKFSDAG QPMSFVLEFH FEPNEYFTNE
251 VLTKTYRMRS EPDDSDPFSF DGPEIMGCTG CQIDWKKGKN VTLKTIKKKQ
301 KHKGRGTVRT VTKTVSNDSF FNFFAPPEVP ESGDLDDDAE AILAADFEIG
351 HFLRERIIPR SVLYFTGEAI EDDDDDYDEE GEEADEGYQL FEEVKSCSKL
401 FQRWLQ
HITS AT: 169-174

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:306971

REFERENCE 2: 133:233618

L6 ANSWER 20 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **223503-67-1** REGISTRY
CN Protein NAP1 (nucleosome assembly protein 1) (rat clone T23 reduced) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Nucleosome assembly protein 1 (rat clone T23 reduced)
CI MAN
SQL 390

SEQ
1 MADIDNKEQS ELDQDLEDVE EVEEEETGEE TKIKARQLTV QMMQNPQILA
51 ALQERLDGLV DTPTGYIESL PKVVKRRVNA LKNLQVKCAQ IEAKFYEEVH
101 DLERKYAVLY QPLFDKRFEI INAIYEPTEE ECEWKPDEED EVSEELKEKA
151 KIEDEKKDEE KEDPKGIPEF WLTVFKNDLL SDMVQEHDEP ILKHLKDIKV
201 KFSDAGQPMS FILEFHFEPN EYFTNEVLTK TYRMRSEPDD SDPFSFDGPE
251 IMGCTGCQID WKKGKNVTLK TIKKKQKHKG RGTVRTVTKT VSKTSFFNFF
301 APPEVPENGD LDDDXEAILA ADFEIGHFLR ERIIPRSVLY FTGEAIEDDD
351 DDYDEEGEEA DEEGEEEGDE ENDPDYDPKK DQNPAECKQQ
HITS AT: 146-151

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 130:308093

L6 ANSWER 21 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **208408-72-4** REGISTRY
CN Protein (Streptococcus pneumoniae clone 3175860) (9CI) (CA INDEX NAME)
CI MAN
SQL 66

SEQ
1 VILEGNYRAT AGREEMKEAI LEYQANPAAL KDLKEKAKNI SREYSEEHLL
51 QIWLDFYEKQ AALGTK
HITS AT: 33-38

REFERENCE 1: 129:40139

L6 ANSWER 22 OF 22 REGISTRY COPYRIGHT 2004 ACS on STN
RN **186984-83-8** REGISTRY
CN Protein (Helicobacter pylori strain J99 open reading frame 05cp11911orf15) (9CI) (CA INDEX NAME)
CI MAN
SQL 237

SEQ
1 GLNYIDLALL VVVVAFGIRG FYHGFVSEIA ATLGIVLGVY LASRYSVAVG
51 NLFSEHLYDL RNETMTNLIG FLLVLASIWV FFLALGVLLG KMLVFSGLGI
101 IDKALGFIFS CLKTFLVLSF ILYALSKMDL MKDANAYLQE KSAIFPTMKS
151 VASKIMRLDG VKHVEKNLKD NLEEMSDEVK NKGSIDNAKE SFNKATDKGV

201 EALKEKAKDL PKNMLEPKHN KPNQTPPIPT PSNKEPL
======
HITS AT: 203-208

REFERENCE 1: 126:176879

FILE 'REGISTRY' ENTERED AT 08:57:39 ON 23 APR 2004
L7 2 S (FP3 OR FP4 OR FP5 OR FP6 OR FP7 OR FP8 OR FP10)/CN

-key terms omitted FP9; See L24-L31

FILE 'HCAPLUS' ENTERED AT 08:57:44 ON 23 APR 2004
L7 2 SEA FILE=REGISTRY ABB=ON PLU=ON (FP3 OR FP4 OR FP5 OR FP6 OR FP7 OR FP8 OR FP10)/CN
L8 476 SEA FILE=HCAPLUS ABB=ON PLU=ON L7 OR FP3 OR FP4 OR FP5 OR FP6 OR FP7 OR FP8 OR FP10 OR FP(W)(3 OR 4 OR 5 OR 6 OR 7 OR 8 OR 10)
L9 9 SEA FILE=HCAPLUS ABB=ON PLU=ON L8 AND RECOMBINAN?

L10 9 L9 NOT L4

L10 ANSWER 1 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 28 Sep 2003
ACCESSION NUMBER: 2003:758741 HCAPLUS
DOCUMENT NUMBER: 140:26952
TITLE: Fusion protein of interleukin-6 and interleukin-6 receptor without a polypeptide linker
AUTHOR(S): Yasukawa, Kiyoshi; Tsuchiya, Shigeo; Ekida, Teiji; Iida, Hiroshi; Ide, Teruhiko; Miki, Daisuke; Yagame, Harutaka; Murayama, Keiichi; Ishiguro, Takahiko
CORPORATE SOURCE: Tokyo Research Laboratories, Tosoh Corporation, Kanagawa, 252-1123, Japan
SOURCE: Journal of Bioscience and Bioengineering (2003), 96(1), 38-46
CODEN: JBBIF6; ISSN: 1389-1723
PUBLISHER: Society for Biotechnology, Japan
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **FP6**, a novel **recombinant** fusion protein of interleukin-6 (IL-6) and IL-6 receptor (IL-6R), was prepared in the methylotrophic yeast Pichia pastoris. This protein was a potent activator of a cell surface transducing glycoprotein, gp130 and is a potential therapeutical reagent in the hemopoietic field. A linker is generally thought to be required for two fused mols. to retain their proper structures although it should preferably be removed to reduce possible antigenicity. It was found that the C-terminal residue of IL-6R could be directly linked to the N-terminal residue of IL-6 without decreasing the ability of IL-6 to bind gp130 and send the IL-6 signal. It was also found that the peptide bond between Lys-37 and Asp-38 of IL-6 was prone to proteolytic cleavage and that the Ig (Ig)-like region of IL-6R underwent extensive and heterogeneous glycosylation when expressed in P. pastoris. Based on these findings, we designed **FP6** without the Ig-like region, in which the C-terminal residue of Ala-333 of IL-6R was directly linked to Asp-38 of IL-6 by a peptide bond. Purified

FP6 had both an in vitro effect on hemopoietic progenitors to generate various colonies and an in vivo effect on megakaryocyte progenitors to increase platelet counts. Four purified FP6s were obtained, which had the same mol. mass and different isoelec. points without any detectable modification in the course of purification The difference in isoelec. points was shown to be due to microheterogeneity of the carbohydrate chains. Each **FP6** had the same specific activity in the cell growth assay with or without endoglycosidase digestion. Homogeneous **FP6** with respect to isoelec. point as well as mol. mass merits more detailed characterization and evaluation for possible clin. application.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 06 Dec 2002
ACCESSION NUMBER: 2002:927445 HCAPLUS
DOCUMENT NUMBER: 138:21184
TITLE: Kindling fluorescent proteins from Anthozoa and Heteractis crispa and their mutants and methods for their use
INVENTOR(S): Lukyanov, Sergey Anatolievich; Chudakov, Dmitry; Lukyanov, Konstantin
PATENT ASSIGNEE(S): Clontech Laboratories, Inc., USA
SOURCE: PCT Int. Appl., 96 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 16
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002096924	A1	20021205	WO 2002-US16379	20020524
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1390379	A1	20040225	EP 2002-746443	20020524
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.:
US 2001-293752P P 20010525
US 2001-329176P P 20011011
WO 2002-US16379 W 20020524

AB Kindling fluorescent protein (KFP) compns. and nucleic acids encoding the same, as well as methods for using the same, are provided. In particular, protein FP595 from Anthozoa (also called AsFP595, or **FP7**, or KFP04) and its two mutants (A148G, and

F90L-A148G-H203Y resp.), and another Heteractis crispa chromoprotein **FP10** (KFP08) and its four mutants (a:K28M-N165A, b:K28M-N165G, c:G20C-T39A-L126H-C148A-N165G-R176H-L181H-A190V-I203H-P208L-K211E, and d:T39A-C148S-N165S-L181H-1203H-P208R-K211E resp.) are provided. These wild-type or mutant kindling fluorescent proteins are expressed **recombinantly** (as his6 epitope tagged fusion proteins) and purified for further characterization. In general, they become brightly fluorescent proteins, from an initial non-fluorescent or low fluorescent state, upon exposure to a kindling stimulus, which fluorescent state may be reversible or irreversible. Specifically, their kindling wavelength of said kindling stimulus ranges from about 200 to 1500 nm, their kindling stimulus ranges from about 0.01 to about 106 W/cm2, and their kindling duration of said kindling stimulus ranges from about 1 ms to about 60 min. The subject protein/nucleic acid compns. find use in labeling protocols, e.g., in labeling proteins, organelles, cells and organisms, etc., in a variety of different types of applications. Also provided are systems and kits for use in practicing such applications. The use of a KFP to study cell migration during embryogenesis, and to study migration of a mitochondrion are demonstrated.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 27 Jul 2001
ACCESSION NUMBER: 2001:543149 HCAPLUS
DOCUMENT NUMBER: 135:255534
TITLE: Role of α2-macroglobulin in regulating amyloid β-protein neurotoxicity: protective or detrimental factor?
AUTHOR(S): Fabrizi, Cinzia; Businaro, Rita; Lauro, Giuliana M.; Fumagalli, Lorenzo
CORPORATE SOURCE: Department of Biology, University "Roma Tre", Rome, 00146, Italy
SOURCE: Journal of Neurochemistry (2001), 78(2), 406-412
CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB α2-Macroglobulin (α2M) has been identified as a carrier protein for β-amyloid (Aβ) decreasing fibril formation and affecting the neurotoxicity of this peptide. The α2-macroglobulin receptor/low d. lipoprotein receptor related protein (LRP) is involved in the internalization and degradation of the α2M/Aβ complexes and its impairment has been reported to occur in Alzheimer's disease. Previous studies have shown α2M to determine an enhancement or a reduction of Aβ toxicity in different culture systems. In order to clarify the role of α2M in Aβ neurotoxicity, we challenged human neuroblastoma cell lines with activated α2M in combination with Aβ. Our results show that in neuroblastoma cells expressing high levels of LRP, the administration of activated α2M protects the cells from Aβ neurotoxicity. Conversely, when this receptor is not present α2M dets. an increase in Aβ toxicity as evaluated

by MTT and TUNEL assays. In LRP-neg. cells transfected with the full-length human LRP, the addition of activated α2M resulted to be protective against Aβ-induced neurotoxicity. By means of **recombinant** proteins we ascribed the neurotoxic activity of α2M to its **FP3** fragment which has been previously shown to bind and neutralize transforming growth factor-β. These studies provide evidence for both a neuroprotective and neurotoxic role of α2M regulated by the expression of its receptor LRP.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 18 Apr 2001
ACCESSION NUMBER: 2001:271410 HCAPLUS
DOCUMENT NUMBER: 135:57730
TITLE: Stable expression of the human 5α-reductase isoenzymes type I and type II in HEK293 cells to identify dual and selective inhibitors
AUTHOR(S): Reichert, Wolfgang; Hartmann, Rolf W.; Jose, Joachim
CORPORATE SOURCE: Fachrichtung 12.1 Pharmazeutische und Medizinische Chemie, Universitat des Saarlandes, Saarbrucken, D-66041, Germany
SOURCE: Journal of Enzyme Inhibition (2001), 16(1), 47-53
CODEN: ENINEG; ISSN: 8755-5093
PUBLISHER: Harwood Academic Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A eucaryotic cell assay was established to identify novel, dual and selective inhibitors of human 5α-reductase. For this purpose the cDNAs encoding 5α-reductase type I and type II were inserted into a pRcCMV vector and expressed in human embryonic kidney (HEK293) cells. Single cell clones with substantially high enzymic activity were selected and established as permanent cell lines. KM values were determined for both isoenzymes. The inhibitory potency of several steroidal and non-steroidal compds. synthesized in our group, as well as finasteride and 4MA as controls, were tested by measuring the conversion of [3H]androstenedione. Reaction products were quantified by a HPLC reversed phase technique. Using the new cell assays, selective as well as novel dual 5α-reductase inhibitors with IC50 values between 1.0 and 2.5μM were identified.

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 05 Feb 1994
ACCESSION NUMBER: 1994:47392 HCAPLUS
DOCUMENT NUMBER: 120:47392
TITLE: Cloning and expression of vertebrate F-spondin cDNA and uses of F-spondin

INVENTOR(S): Jessell, Thomas M.; Klar, Avihu
PATENT ASSIGNEE(S): Columbia University, USA
SOURCE: PCT Int. Appl., 102 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9320196	A1	19931014	WO 1993-US3164	19930402
W: AU, BB, BG, BR, CA, FI, HU, JP, KP, KR, LK, MG, MN, MW, NO, NZ, PL, RO, RU, SD, SE, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5279966	A	19940118	US 1992-862021	19920402
ZA 9302362	A	19940615	ZA 1993-2362	19930401
AU 9339455	A1	19931108	AU 1993-39455	19930402
AU 677185	B2	19970417		
EP 670895	A1	19950913	EP 1993-908743	19930402
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 07508402	T2	19950921	JP 1993-517749	19930402
US 5750502	A	19980512	US 1995-313288	19950105
AU 9712698	A1	19970515	AU 1997-12698	19970213
AU 713198	B2	19991125		

PRIORITY APPLN. INFO.: US 1992-862021 19920402
WO 1993-US3164 19930402

AB Vertebrate nucleic acid encoding F-spondin, expression of this nucleic acid in **recombinant** cells to produce F-spondin, and use of F-spondin for attachment of nerve cells to a matrix or to stimulate the growth or regeneration of nerve cells are claimed. Subtractive hybridization techniques were used to isolate cDNA expressed selectively by the floor plate. A rat cDNA clone encoding a novel secreted protein (F-spondin) that is expressed at high levels by the rat floor plate during embryonic development was isolated and sequenced. The predicted amino acid sequence of F-spondin indicates the protein contains domains similar to those in thrombospondin and other proteins implicated in cell adhesion and neurite outgrowth. In vitro assays indicated that F-spondin promotes neural cell adhesion and neurite outgrowth. The cDNAs for chicken and Xenopus F-spondin were also cloned.

L10 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 13 Jun 1992
ACCESSION NUMBER: 1992:229240 HCAPLUS
DOCUMENT NUMBER: 116:229240
TITLE: Cloning of the nifA and nifB genes of Azospirillum brasilense strain FP2
AUTHOR(S): Knopik, M. A.; Funayama, S.; Rigo, L. U.; Souza, E. M.; Machado, H. B.; Pedrosa, F. O.
CORPORATE SOURCE: Dep. Biochem., Univ. Fed. Parana, Curitiba, 81504, Brazil
SOURCE: Developments in Plant and Soil Sciences (1991), 48(Nitrogen Fixation), 133-8

CODEN: DVPSD8; ISSN: 0167-840X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A genomic library of Azospirillum brasilense was constructed and used to isolate the nifA gene by complementation of a nifA- mutant of A. brasilense (**FP10**). A **recombinant** plasmid, pMAK7, was isolated and found to contain a functional nifA gene and to hybridize with nifA and nifB probes from Herbaspirillum seropedicae.

L10 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 16 May 1992
ACCESSION NUMBER: 1992:190529 HCAPLUS
DOCUMENT NUMBER: 116:190529
TITLE: Diagnostic value of a synthetic peptide derived from Echinococcus granulosus **recombinant** protein
AUTHOR(S): Chamekh, M.; Gras-Masse, H.; Bossus, M.; Facon, B.; Dissous, C.; Tartar, A.; Capron, A.
CORPORATE SOURCE: Cent. Immunol. Biol. Parasit., Inst. Pasteur, Lille, 59 019, Fr.
SOURCE: Journal of Clinical Investigation (1992), 89(2), 458-64
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A specific monoclonal antibody (MAb; EG 02 154/12) directed against a protein epitope of E. granulosus antigen 5 was used to screen a cDNA library constructed from E. granulosus protoscoleces RNA. One clone designated Eg14 was selected and shown to code for an amino acid sequence partially homologous to that of the clone Eg6 previously identified with the same MAb. Hydrophobic cluster anal. showed that both **recombinant** antigens may adopt a similar α-helical organization and share a common conformational epitope. A synthetic peptide (89-122) mimicking the conformational site of Eg6 and Eg14 was constructed and demonstrated to be able to inhibit binding of the MAb and human hydatid sera to the Eg6 fusion protein (**FP6**) or to native hydatid antigens. To assess the diagnostic value of the peptide 89-122, the authors tested sera from patients infected with different parasites for their antibody reactivity with this peptide in ELISA. A high binding sensitivity and specificity of IgG-A-M antibodies were obtained with E. granulosus-infected patient sera. Moreover, the peptide 89-122 was specifically recognized by IgE antibodies from patients with hydatid disease. These results indicate the particular interest of this synthetic peptide as a standardized antigen in diagnosis and treatment surveillance of hydatidosis.

L10 ANSWER 8 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 18 Oct 1991
ACCESSION NUMBER: 1991:552401 HCAPLUS
DOCUMENT NUMBER: 115:152401
TITLE: Cloning and characterization of the nifA gene from Herbaspirillum seropedicae strain Z78
AUTHOR(S): Souza, Emanuel M.; Funayama, Shigehiro; Rigo, Liu U.; Pedrosa, Fabio O.

CORPORATE SOURCE: Dep. Biochem., Univ. Fed. Parana, Curitiba, 81504, Brazil
SOURCE: Canadian Journal of Microbiology (1991), 37(6), 425-9
CODEN: CJMIAZ; ISSN: 0008-4166
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A genomic library of H. seropedicae was constructed and screened for the nifA gene by complementation of a nifA mutant of Azospirillum brasilense (**FP10**). A **recombinant** plasmid, pEMS1, capable of restoring acetylene reduction activity in the mutant **FP10**, was isolated and found to hybridize to the nifA gene of Klebsiella pneumoniae. The results suggest that nifA is involved in the regulation of nif genes in H. seropedicae.

L10 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 21 Jan 1990
ACCESSION NUMBER: 1990:17367 HCAPLUS
DOCUMENT NUMBER: 112:17367
TITLE: **Recombinant** avipox virus for expression of non-avipox DNA, especially avian or mammalian pathogen-encoding DNA
INVENTOR(S): Paoletti, Enzo
PATENT ASSIGNEE(S): Health Research, Inc., USA
SOURCE: PCT Int. Appl., 89 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 8903429	A1	19890420	WO 1988-US2816	19880824
W: AT, AU, BG, BR, CH, DE, DK, GB, HU, JP, KP, KR, LU, NL, NO, SE, SU				
JP 2002186494	A2	20020702	JP 2001-311076	19870828
AU 8824275	A1	19890502	AU 1988-24275	19880824
NL 8820679	A	19890703	NL 1988-20679	19880824
NL 195051	C	20030701		
DE 3890874	T	19891221	DE 1988-3890874	19880824
DE 3890874	C2	20030313		
JP 02500879	T2	19900329	JP 1988-507715	19880824
JP 3348156	B2	20021120		
CH 679933	A	19920515	CH 1989-1652	19880824
CH 679934	A	19920515	CH 1991-1444	19880824
AT 8809007	A	19950515	AT 1988-9007	19880824
AT 408549	B	20011227		
JP 2002348255	A2	20021204	JP 2002-71416	19880824
FR 2621487	A1	19890414	FR 1988-11334	19880829
FR 2621487	B1	19911018		
ZA 8806415	A	19890426	ZA 1988-6415	19880829
BE 1002134	A5	19900724	BE 1988-978	19880829
GB 2217718	A1	19891101	GB 1989-8921	19890420
GB 2217718	B2	19920520		
DK 8902036	A	19890627	DK 1989-2036	19890427

KR 9711149	B1	19970707	KR 1989-70753	19890428
AU 9516288	A1	19950817	AU 1995-16288	19950405
AU 690210	B2	19980423		
AU 9877412	A1	19981008	AU 1998-77412	19980721
AU 725985	B2	20001026		
AU 9931252	A1	19990916	AU 1999-31252	19990525
AU 747139	B2	20020509		
AU 770916	B2	20040304	AU 2000-59413	20000913
AU 769221	B2	20040122	AU 2000-72198	20001212
AU 2000072198	A5	20010222		
AU 761321	B2	20030605	AU 2001-16636	20010125

PRIORITY APPLN. INFO.:

US 1987-90711	A	19870828
US 1987-110335	A	19871020
US 1988-186054	A	19880425
US 1988-234390	A	19880823
JP 1988-507715	A3	19870828
CH 1989-1652	A	19880824
WO 1988-US2816	A	19880824
AU 1992-15871	A0	19920309
AU 1995-22755	A3	19950406
AU 1996-61601	A3	19960606
AU 1997-12780	A3	19961202
AU 1998-77412	A3	19980721

AB A method for expressing a pathogen-encoding gene in a vertebrate to induce an immunol. response to the pathogen comprises inoculating the vertebrate with a **recombinant** virus, e.g. avipoxvirus, containing a DNA insertion in a non-essential region of the genome, and expressing the DNA without productive replication of the virus in the vertebrate. Plasmid pRW 735.1 containing an insertion of Pi promoter, rabies G antigen gene, 11K promoter, and lac Z in non-replicable genome of the fowl pox virus was constructed and used to prepare **recombinant** fowlpox virus vFP-2 by cotransfection with fowlpox virus **FP-5** into chicken embryo fibroblast cells. Rabies antigen gene was expressed on the surface of avian and non-avian cells infected with vFP-2 virus. Rabbits inoculated intradermally with vFP-2 virus produced typical pox lesions, and detectable anti-β-galactosidase and anti-rabies antibodies.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 08:59:08 ON 23 APR 2004)

L11 37 S L9
L12 26 DUP REM L11 (11 DUPLICATES REMOVED)

L12 ANSWER 1 OF 26 MEDLINE on STN
ACCESSION NUMBER: 2003233572 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12755614
TITLE: Growth factor-binding sequence in human alpha2-macroglobulin targets the receptor-binding site in transforming growth factor-beta.
AUTHOR: Arandjelovic Sanja; Freed Tiffany A; Gonias Steven L
CORPORATE SOURCE: Department of Biochemistry and Molecular Genetics, Box 800214, Charlottesville, Virginia 22908, USA.
CONTRACT NUMBER: CA-53462 (NCI)
SOURCE: Biochemistry, (2003 May 27) 42 (20) 6121-7. Journal code: 0370623. ISSN: 0006-2960.

Searcher : Shears 571-272-2528

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20030521
Last Updated on STN: 20030703
Entered Medline: 20030702

AB alpha(2)-Macroglobulin (alpha(2)M) binds transforming growth factor-beta1 (TGF-beta1) and TGF-beta2, forcing these growth factors into a state of latency. The mechanism by which this occurs remains unclear. In this paper, we demonstrate that peptides, derived from the structure of human alpha(2)M (amino acids 714-729), bind directly to TGF-beta1 and block the binding of TGF-beta1 to the type I and II TGF-beta receptors. The alpha(2)M-derived peptides are notable for hydrophobic tripeptide sequences (WIW or VVV) and acidic residues (Glu(714) and Asp(719) in the mature alpha(2)M subunit), which may function analogously to the structural elements that mediate TGF-beta-binding in the type II receptor. Mutating Glu(714) and Asp(719) in the alpha(2)M-peptide-GST fusion protein, **FP3**, which contains the putative growth factor-binding site, significantly decreased the binding affinity of **FP3** for TGF-beta1. The alpha(2)M-derived peptides, which bind TGF-beta1, inhibited the interaction of TGF-beta1 with its receptors in fetal bovine heart endothelial cells. The same peptides also inhibited the activity of TGF-beta1 in endothelial cell proliferation assays. These results demonstrate that alpha(2)M-derived peptides target the receptor-binding sequence in TGF-beta.

L12 ANSWER 2 OF 26 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 1

ACCESSION NUMBER: 2003:552461 BIOSIS
DOCUMENT NUMBER: PREV200300555346
TITLE: Fusion protein of interleukin-6 and interleukin-6 receptor without a polypeptide linker.
AUTHOR(S): Yasukawa, Kiyoshi [Reprint Author]; Tsuchiya, Shigeo; Ekida, Teiji; Iida, Hiroshi; Ide, Teruhiko; Miki, Daisuke; Yagame, Harutaka; Murayama, Keiichi; Ishiguro, Takahiko
CORPORATE SOURCE: Tokyo Research Laboratories, Tosoh Corporation, 2743-1 Hayakawa, Ayase-shi, Kanagawa, 252-1123, Japan yasukawa@tosoh.co.jp
SOURCE: Journal of Bioscience and Bioengineering, (July 2003) Vol. 96, No. 1, pp. 38-46. print.
ISSN: 1389-1723.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 26 Nov 2003
Last Updated on STN: 26 Nov 2003

AB **FP6**, a novel **recombinant** fusion protein of interleukin-6 (IL-6) and IL-6 receptor (IL-6R), was prepared in the methylotrophic yeast Pichia pastoris. This protein was a potent activator of a cell surface transducing glycoprotein, gp130 and is a potential therapeutical reagent in the hemopoietic field. A linker is generally thought to be required for two fused molecules to retain their proper structures although it should preferably be

removed to reduce possible antigenicity. It was found that the C-terminal residue of IL-6R could be directly linked to the N-terminal residue of IL-6 without decreasing the ability of IL-6 to bind gp130 and send the IL-6 signal. It was also found that the peptide bond between Lys-37 and Asp-38 of IL-6 was prone to proteolytic cleavage and that the immunoglobulin (Ig)-like region of IL-6R underwent extensive and heterogeneous glycosylation when expressed in P. pastoris. Based on these findings, we designed **FP6** without the Ig-like region, in which the C-terminal residue of Ala-333 of IL-6R was directly linked to Asp-38 of IL-6 by a peptide bond. Purified **FP6** had both an in vitro effect on hemopoietic progenitors to generate various colonies and an in vivo effect on megakaryocyte progenitors to increase platelet counts. Four purified FP6s were obtained, which had the same molecular mass and different isoelectric points without any detectable modification in the course of purification. The difference in isoelectric points was shown to be due to microheterogeneity of the carbohydrate chains. Each **FP6** had the same specific activity in the cell growth assay with or without endoglycosidase digestion. Homogeneous **FP6** with respect to isoelectric point as well as molecular mass merits more detailed characterization and evaluation for possible clinical application.

L12 ANSWER 3 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-129517 [17] WPIDS
DOC. NO. CPI: C2002-039638
TITLE: Novel peptide derived from alpha2-macroglobulin that binds and inhibits transforming growth factor activity useful for treating rheumatoid arthritis, psoriasis, atherosclerosis, liver fibrosis and cancer.
DERWENT CLASS: B04
INVENTOR(S): GONIAS, S L; WEBB, D J
PATENT ASSIGNEE(S): (UYVI-N) UNIV VIRGINIA PATENT FOUND
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 6329500	B1	20011211	(200217)*		21

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 6329500	B1 Provisional	US 1998-85574P	19980515
		US 1999-311352	19990513

PRIORITY APPLN. INFO: US 1998-85574P 19980515; US 1999-311352 19990513
AN 2002-129517 [17] WPIDS
AB US 6329500 B UPAB: 20020313
NOVELTY - A substantially pure peptide (I) derived from alpha 2-macroglobulin or peptide fragment consisting of a sequence (S1) of

113 amino acids defined in the specification or its fragment, which retains the ability to bind to transforming growth factor- beta (TGF- beta), is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a composition (II) for inhibiting TGF- beta activity, comprising (I).

ACTIVITY - Antirheumatic; Antiarthritic; Antiinflammatory; Antipsoriatic; Antiatherosclerotic; Nephrotropic; Cytostatic.

MECHANISM OF ACTION - Inhibitor of TGF- beta activity.

The peptide fragment **FP3**, isolated from the structure of alpha 2-macroglobulin (alpha 2M), contained the binding site for transforming growth factor- beta 1 (TGF- beta 1) and TGF- beta 2. To demonstrate that 125I-TGF- beta binding to **FP3** was non-covalent and specific, 125I-TGF- beta was incubated with polyvinylidene difluoride (PVDF)-immobilized **FP3** in the presence of excess solution-phase **FP3** or unlabeled TGF- beta . **FP3** (1 micro M) in solution inhibited the binding of 125I-TGF- beta 1 and 125I-TGF- beta 2 to immobilized **FP3** by 94 plus or minus 3% and 92 plus or minus 5%, respectively. Unlabeled TGF- beta 1 (0.2 micro M) inhibited 125I-TGF- beta 1 binding to immobilized **FP3** by 72 plus or minus 8%, and unlabeled TGF- beta 2 (0.2 micro M) inhibited 125I-TGF- beta 2 binding to immobilized **FP3** by 90 plus or minus 4%. **FP3** not only bound TGF- beta 1 and TGF- beta 2, but also neutralized the activities of these cytokines. When added to RAW 264.7 cell cultures, **FP3** promoted the accumulation of nitrite more effectively than methylamine-modified alpha 2M (alpha 2M-MA). Since nitric oxide (NO) synthesis by alpha 2M was due to the neutralization of TGF- beta , the increased potency of **FP3** was due to its increased binding affinity for TGF- beta . To test this hypothesis, the secretion of TGF- beta 1 and TGF- beta 2 by RAW 264.7 cells was measured using isoform-specific enzyme linked immunosorbent assays (ELISAs). In medium which was conditioned for 24 hours, the concentrations of active and total (active+latent) TGF- beta 1 were 2 and 10 pM, respectively. The concentrations of active and total TGF- beta 2 were 1 and 4 pM, respectively. The ELISA studies confirmed that RAW 264.7 cells expressed both TGF- beta isoforms but higher levels of TGF- beta 1, supporting the hypothesis that the increased potency of **FP3** reflected its increased capacity to neutralize TGF- beta 1.

USE - (I) is useful for inhibiting TGF- beta activity in vivo and treating a pathologic condition caused by or resulting from TGF- beta activity, including inflammation, for e.g. rheumatoid arthritis, inflamed skin lesions, scar tissue formation, lung fibrosis, liver fibrosis, atherosclerosis, cancer, psoriasis or glomerulonephritis. (I) prevents or inhibits TGF- beta activity including stimulation of cell proliferation and stimulation of inflammation, inhibition of the immune response and promotion of extracellular matrix proteins. (II) is useful for inhibiting cytokine activity in a patient.

Dwg.1/8

L12 ANSWER 4 OF 26 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001412233 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11461976
TITLE: Role of alpha2-macroglobulin in regulating amyloid

beta-protein neurotoxicity: protective or detrimental factor?.
AUTHOR: Fabrizi C; Businaro R; Lauro G M; Fumagalli L
CORPORATE SOURCE: Department of Biology, University 'Roma Tre', Rome, Italy.. fabrizi@uniroma3.it
SOURCE: Journal of neurochemistry, (2001 Jul) 78 (2) 406-12. Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010827
Last Updated on STN: 20010827
Entered Medline: 20010823

AB alpha2-Macroglobulin (alpha2M) has been identified as a carrier protein for beta-amyloid (Abeta) decreasing fibril formation and affecting the neurotoxicity of this peptide. The alpha2-macroglobulin receptor/low density lipoprotein receptor related protein (LRP) is involved in the internalization and degradation of the alpha2M/Abeta complexes and its impairment has been reported to occur in Alzheimer's disease. Previous studies have shown alpha2M to determine an enhancement or a reduction of Abeta toxicity in different culture systems. In order to clarify the role of alpha2M in Abeta neurotoxicity, we challenged human neuroblastoma cell lines with activated alpha2M in combination with Abeta. Our results show that in neuroblastoma cells expressing high levels of LRP, the administration of activated alpha2M protects the cells from Abeta neurotoxicity. Conversely, when this receptor is not present alpha2M determines an increase in Abeta toxicity as evaluated by MTT and TUNEL assays. In LRP-negative cells transfected with the full-length human LRP, the addition of activated alpha2M resulted to be protective against Abeta-induced neurotoxicity. By means of **recombinant** proteins we ascribed the neurotoxic activity of alpha2M to its **FP3** fragment which has been previously shown to bind and neutralize transforming growth factor-beta. These studies provide evidence for both a neuroprotective and neurotoxic role of alpha2M regulated by the expression of its receptor LRP.

L12 ANSWER 5 OF 26 MEDLINE on STN
ACCESSION NUMBER: 2001306747 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11243705
TITLE: Directly linked soluble IL-6 receptor-IL-6 fusion protein induces astrocyte differentiation from neuroepithelial cells via activation of STAT3.
AUTHOR: Takizawa T; Yanagisawa M; Ochiai W; Yasukawa K; Ishiguro T; Nakashima K; Taga T
CORPORATE SOURCE: Department of Cell Fate Modulation, Institute of Molecular Embryology and Genetics, Kumamoto University, Kumamoto, Japan.
SOURCE: Cytokine, (2001 Mar 7) 13 (5) 272-9. Journal code: 9005353. ISSN: 1043-4666.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010604
Last Updated on STN: 20010604
Entered Medline: 20010531

AB Signals of interleukin 6 (IL-6) are transduced by binding of IL-6 to its cell surface receptor (IL-6R) and subsequent association of the resultant IL-6/IL-6R complex with gp130, the signal transducing receptor component utilized in common by all the IL-6 family of cytokines. A soluble form of IL-6R (sIL-6R), which lacks transmembrane and cytoplasmic regions, retains the ability to bind IL-6 and signal through gp130. We show here that a fusion protein of sIL-6R and IL-6 without a polypeptide linker, termed **FP6**, induces differentiation of astrocytes from fetal mouse neuroepithelial cells as potently as a representative IL-6 family cytokine, leukaemia inhibitory factor (LIF). **FP6** has a potential to activate a transcription factor, signal transducer and activator of transcription 3 (STAT3), and mitogen-activated protein kinases, ERK1 and ERK2, in these cells as does LIF. **FP6** activates a promoter of the gene for an astrocytic marker, glial fibrillary acidic protein (GFAP), in neuroepithelial cells. This activation is virtually abolished by ectopic expression of a dominant-negative form of STAT3, or by introducing a point mutation into the STAT3 response element located in the GFAP promoter. These results suggest that **FP6** induces astrocyte differentiation from neuroepithelial cells through STAT3 activation and that **FP6** could be of use as a substitute for natural IL-6 family cytokines.

L12 ANSWER 6 OF 26 MEDLINE on STN
ACCESSION NUMBER: 2000148793 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10681572
TITLE: Identical or overlapping sequences in the primary structure of human alpha(2)-macroglobulin are responsible for the binding of nerve growth factor-beta, platelet-derived growth factor-BB, and transforming growth factor-beta.
AUTHOR: Gonias S L; Carmichael A; Mettenburg J M; Roadcap D W; Irvin W P; Webb D J
CORPORATE SOURCE: Department of Pathology, University of Virginia Health Sciences Center, Charlottesville, Virginia 22908, USA.. slg2t@virginia.edu
CONTRACT NUMBER: CA-53462 (NCI)
SOURCE: Journal of biological chemistry, (2000 Feb 25) 275 (8) 5826-31.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000407
Last Updated on STN: 20000407
Entered Medline: 20000330

AB alpha(2)-Macroglobulin (alpha(2)M) functions as a proteinase inhibitor and as a carrier of diverse growth factors. In this

study, we localized binding sites for platelet-derived growth factor-BB (PDGF-BB) and nerve growth factor-beta (NGF-beta) to a linear sequence in the 180-kDa human alpha(2)M subunit which includes amino acids 591-774. A glutathione S-transferase fusion protein containing amino acids 591-774 (**FP3**) bound PDGF-BB and NGF-beta in ligand blotting assays whereas five other fusion proteins, which collectively include amino acids 99-590 and 775-1451 did not. The K(D) values for PDGF-BB and NGF-beta binding to immobilized **FP3** were 300 +/- 40 and 180 +/- 30 nM, respectively; these values were comparable with those determined using methylamine-modified alpha(2)M, suggesting that higher-order alpha(2)M structure is not necessary for PDGF-BB and NGF-beta binding. PDGF-BB and NGF-beta blocked the binding of transforming growth factor-beta1 (TGF-beta1) to **FP3**. Furthermore, murinoglobulin, which is the only known member of the alpha-macroglobulin family that does not bind TGF-beta, also failed to bind PDGF-BB and NGF-beta. These results support the hypothesis that either a single linear sequence in human alpha(2)M or overlapping sequences are responsible for the binding of TGF-beta, PDGF-BB, and NGF-beta, even though there is minimal sequence identity between these three growth factors. **FP3** blocked the binding of PDGF-BB to a purified chimeric protein, in which the extracellular domain of the PDGF beta receptor was fused to the IgG(1) Fc domain, and to PDGF receptors on NIH 3T3 cells. Thus, **FP3** may inhibit the activity of PDGF-BB.

L12 ANSWER 7 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1999-153770 [13] WPIDS
CROSS REFERENCE: 1997-393685 [36]
DOC. NO. CPI: C1999-045497
TITLE: Fusion and chimaeric proteins including cyclin-dependent kinase binding motif - used for regulation of cell proliferation and differentiation, for treatment of, e.g. vascular injury, cancers, fibrosis and neurodegeneration.
DERWENT CLASS: B04 D16
INVENTOR(S): BEACH, D H; GYURIS, J; LAMPHERE, L
PATENT ASSIGNEE(S): (MITO-N) MITOTIX INC; (BEAC-I) BEACH D H; (GYUR-I) GYURIS J; (LAMP-I) LAMPHERE L; (GPCB-N) GPC BIOTECH INC
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9906540	A2	19990211	(199913)*	EN	87
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9886014	A	19990222	(199927)		
EP 1000166	A2	20000517	(200028)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2001512008	W	20010821	(200155)		116

US 2002068706 A1 20020606 (200241)
AU 2002027625 A 20020516 (200244)#
AU 752152 B 20020905 (200264)
US 6495526 B2 20021217 (200307)

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9906540	A2		WO 1998-US15759	19980729
AU 9886014	A		AU 1998-86014	19980729
EP 1000166	A2		EP 1998-937264	19980729
			WO 1998-US15759	19980729
JP 2001512008	W		WO 1998-US15759	19980729
			JP 2000-505282	19980729
US 2002068706	A1	CIP of	US 1996-589981	19960123
			US 1997-902572	19970729
AU 2002027625	A	Div ex	AU 1998-86014	19980729
			AU 2002-27625	20020322
AU 752152	B		AU 1998-86014	19980729
US 6495526	B2	CIP of	US 1996-589981	19960123
			US 1997-902572	19970729

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9886014	A	Based on	WO 9906540
EP 1000166	A2	Based on	WO 9906540
JP 2001512008	W	Based on	WO 9906540
US 2002068706	A1	CIP of	US 5672508
AU 752152	B	Previous Publ.	AU 9886014
		Based on	WO 9906540
US 6495526	B2	CIP of	US 5672508

PRIORITY APPLN. INFO: US 1997-902572 19970729; US 1996-589981 19960123; AU 2002-27625 20020322

AN 1999-153770 [13] WPIDS
CR 1997-393685 [36]
AB WO 9906540 A UPAB: 20030129

Recombinant transfection system (A) comprises: (i) first gene construct (GC1) comprising a sequence (I) encoding an inhibitory polypeptide (II) containing at least one CDK (cyclin-dependent kinase)-binding motif for binding and inhibiting activity of a CDK, linked to a transcription regulator functional in eukaryotic cells; (ii) second gene construct (GC2) comprising a sequence (III) encoding a polypeptide (IV) that promotes endothelialisation, and (iii) a gene delivery composition (GDC) for delivering the GCs to a cell for transfection. Also new are: (1) nucleic acid (V) encoding a fusion protein (FP) containing: (i) a therapeutic polypeptide sequence (TP) from an intracellular protein that alters a cellular process when FP enters the cell, and (ii) a transcellular polypeptide sequence (TCP) that promotes transcytosis of FP; (2) FP consisting of at least one CDK-binding motif and a TCP; (3) nucleic acid encoding a fusion protein (FP') containing any therapeutic polypeptide sequence and TCP; (4) **FP'**; (

5) nucleic acid (VI) encoding a chimaeric protein (CP) comprising CDK-binding motifs from two or more different proteins; (6) **recombinant** transfection system containing: (i) (VI) linked to a regulator, and (ii) GDC; and (7) CP.

USE - (A) are used to treat vascular wounds that involve a break in the endothelium and excessive proliferation of smooth muscle, particularly restenosis but more generally any repair of cardiovascular damage, arteriosclerotic lesions or for endothelialisation of synthetic vascular grafts. More generally, FP are used to treat unwanted cellular proliferation in a very wide range of situations, e.g. for treating vascular diseases as above; fibrotic disorders (e.g. rheumatoid arthritis, diabetes, cirrhosis); many tumours, e.g. gliomas and leukaemias; chronic inflammation; neurodegeneration; acne; also to control hair growth (e.g. to prevent hair loss caused by chemotherapy or radiation); periodontal disease; to inhibit spermatogenesis; treatment of tachycardia etc. CP bind to CDKs so inhibit cell cycle progression, particularly smooth muscle cell proliferation. The gene constructs may also be used to produce FP in cell cultures, for production or for regulating cell differentiation in vitro.

ADVANTAGE - FP that include an internalisation sequence can be delivered to cells where transfection with a gene construct is ineffective, while those that include TCP represent a general approach to delivery of gene therapy proteins. Specifically TCP ensures that TP is delivered not only to cells actually transfected by the gene therapy vector but also to adjacent cells.

Dwg.0/5

L12 ANSWER 8 OF 26 MEDLINE on STN

ACCESSION NUMBER: 1999360585 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10433203
TITLE: Identification of essential subelements in the hHSD17B1 enhancer: difference in function of the enhancer and that of the hHSD17BP1 analog is due to -480C and -486G.
AUTHOR: Leivonen S; Piao Y S; Peltoketo H; Numchaisrika P; Vihko R; Vihko P
CORPORATE SOURCE: Biocenter Oulu and World Health Organization Collaborating Centre for Research on Reproductive Health, University of Oulu, Finland.
SOURCE: Endocrinology, (1999 Aug) 140 (8) 3478-87. Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990817

AB The function of the gene encoding human 17beta-hydroxysteroid dehydrogenase (17HSD) type 1, the hHSD17B1 gene, is regulated by a cell-specific enhancer at position -662 to -392. The adjacent hHSD17BP1 gene, whose function is not known, contains an analogous region in its 5'-flanking region. The identity between the hHSD17B1 enhancer and the hHSD17BP1 equivalent is as high as 98%, i.e. they

differ by only five nucleotides. Results from reporter gene analyses showed that the hHSD17BP1 analog, a pseudoenhancer, has only 10% the activity of the hHSD17B1 enhancer. Furthermore, the results indicate that the reduced function of the pseudoenhancer is a consequence of the presence of G and A at positions -480 and -486, whereas the hHSD17B1 enhancer contains -480C and -486G. In addition, three protected areas were localized to regions -495/-485 (FP1), -544/-528 (FP2), and -589/-571 (**FP3**) in deoxyribonuclease I footprinting analysis of the hHSD17B1 enhancer. Replacement of the footprinted regions with a nonsense sequence demonstrated that the FP2 region is the most critical for enhancer activity. Mutations of FP2 or a short palindromic region within it led to almost complete abolishment of enhancer activity. We have identified several subelements that are essential for appropriate function of the hHSD17B1 enhancer. The results also show that the hHSD17B1 and hHSD17BP1 genes operate differently despite the high homology between them.

L12 ANSWER 9 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1998-261491 [23] WPIDS
DOC. NO. NON-CPI: N1998-206075
DOC. NO. CPI: C1998-081288
TITLE: New isolated mammalian Fringe gene(s) - used to develop products for treating, e.g. atherosclerosis, angioplasty, cardiovascular disease, angiogenesis or cancer.
DERWENT CLASS: B04 D16 P14
INVENTOR(S): COHEN, B L; EGAN, S E; LIPSHITZ, H D; PHILLIPS, R A
PATENT ASSIGNEE(S): (HSCR-N) HSC RES & DEV LP
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9817793	A1	19980430	(199823)*	EN	89
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: CA JP US					
EP 939814	A1	19990908	(199941)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9817793	A1	WO 1997-CA775	19971020
EP 939814	A1	EP 1997-944671	19971020
		WO 1997-CA775	19971020

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 939814	A1	Based on	WO 9817793

PRIORITY APPLN. INFO: US 1996-28398P 19961021
AN 1998-261491 [23] WPIDS

AB WO 9817793 A UPAB: 19980610
The following are claimed: (1) an isolated nucleic acid comprising a nucleotide sequence (NS) encoding a mammalian Fringe protein (FP); (2) a pure mammalian **FP**; (3) a pure polypeptide comprising at least 15 (especially at least 5) consecutive aa residues of a protein as in (1); (3) an immunogen comprising a portion of a protein as in (2); (4) a **recombinant** vector comprising a nucleic acid as in (4); (5) a host cell comprising a vector as in (4); (6) a purified antibody specific for an antigenic determinant of a mammalian **FP**; (7) a non-human transgenic animal where a genome of the animal, or of an ancestor, has been modified by introduction of a modification selected from: (a) insertion of NS encoding a heterospecific FP; (b) insertion of NS encoding a dominant negative mutant of FP, and (c) inactivation of an endogenous Fringe gene; (8) a method for preventing or treating a disorder in a mammal characterised by an abnormality in a signal transduction pathway comprising an interaction between a Notch receptor and a Notch ligand, comprising modulating the Notch receptor/Notch ligand interaction by administration to the mammal of a mammalian FP or of a fragment or analogue; (9) a method for promoting differentiation of a mammalian cell by suppressing expression of Lunatic FP in the cell and/or promoting expression of Radical FP and/or Manic FP in the cell, and (10) a method for suppressing differentiation of a mammalian cell by suppressing expression of Radical FP and/or Manic FP in the cell and/or promoting expression of Lunatic FP in the cell.

USE - The host cell of may be used to express a mammalian FP (claimed). The FP's control or modulate activation of the Notch receptor by Notch ligands. The Fringe system of proteins can be used to induce new cell fates at tissue boundaries, to reinforce predetermined tissue boundaries and to block Notch signalling in differentiating cells. The products can be used to prepare therapeutics useful to regulate, treat or prevent symptoms related to angioplasty, atherosclerosis, cardiovascular disease or diseases related to angiogenesis, including cancer (claimed). They can also be used to regulate skin growth and differentiation (claimed). The products can also be used for detection, diagnosis and drug screening especially for compounds that modulate expression of a mammalian Fringe gene or that selectively bind to a mammalian FP (claimed).

Dwg.0/9

L12 ANSWER 10 OF 26 MEDLINE on STN
ACCESSION NUMBER: 1999069399 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9852068
TITLE: Hepatocyte nuclear factor-4 controls transcription from a TATA-less human sex hormone-binding globulin gene promoter.
AUTHOR: Janne M; Hammond G L
CORPORATE SOURCE: Department of Obstetrics & Gynecology, and Medical Research Council of Canada Group in Fetal and Neonatal Health and Development, University of Western Ontario, Canada.
SOURCE: Journal of biological chemistry, (1998 Dec 18) 273 (51) 34105-14.
Journal code: 2985121R. ISSN: 0021-9258.

Searcher : Shears 571-272-2528

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M31651
ENTRY MONTH: 199901
ENTRY DATE: Entered STN: 19990209
Last Updated on STN: 19990209
Entered Medline: 19990126

AB Hepatocytes are the major source of sex hormone-binding globulin (SHBG), a glycoprotein that transports sex steroids in the blood and regulates their access to target tissues. The human SHBG proximal promoter was analyzed by DNase I footprinting, and the functional significance of 6 footprinted regions (FP1-**FP6**) within the proximal promoter was studied in human HepG2 hepatoblastoma cells. Two footprinted regions (FP1 and **FP3**) contain binding sites for the chicken ovalbumin upstream promoter-transcription factor (COUP-TF) and hepatocyte nuclear factor-4 (HNF-4). In experiments where SHBG promoter-luciferase reporter gene constructs were co-transfected into HepG2 cells with COUP-TF and/or HNF-4 expression vectors, HNF-4 markedly increased transcription, whereas COUP-TF suppressed this probably by displacing HNF-4 from their common FP1-binding site. This COUP-TF/HNF-4-binding site within FP1 includes a TTTAA sequence, located at nucleotides -30/-26 upstream of the transcription start site, which fails to interact with human TFIID, TATA-binding protein in vitro. When this sequence was replaced with an idealized HNF-4-binding site, the transcriptional activity of the promoter increased in HepG2 cells. Taken together, these data imply that an interplay between COUP-TF and HNF-4 at a site within FP1 regulates human SHBG expression and that HNF-4 controls transcription from this TATA-less promoter by somehow substituting for TATA-binding protein in the recruitment of a transcription preinitiation complex.

L12 ANSWER 11 OF 26 MEDLINE on STN
ACCESSION NUMBER: 1998250795 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9582381
TITLE: Localization of the binding site for transforming growth factor-beta in human alpha2-macroglobulin to a 20-kDa peptide that also contains the bait region.
AUTHOR: Webb D J; Wen J; Karns L R; Kurilla M G; Gonias S L
CORPORATE SOURCE: Department of Pathology, University of Virginia Health Sciences Center, Charlottesville, Virginia 22908, USA.
CONTRACT NUMBER: CA-53462 (NCI)
SOURCE: Journal of biological chemistry, (1998 May 22) 273 (21) 13339-46.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980708
Last Updated on STN: 20021210
Entered Medline: 19980625

AB alpha2-Macroglobulin (alpha2M) functions as a major carrier of transforming growth factor-beta (TGF-beta) in vivo. The goal of this investigation was to characterize the TGF-beta-binding site in alpha2M. Human alpha2M, which was reduced and denatured to generate 180-kDa subunits, bound TGF-beta1, TGF-beta2, and NGF-beta in ligand blotting experiments. Cytokine binding was not detected with bovine serum albumin that had been reduced and alkylated, and only minimal binding was detected with purified murinoglobulin. To localize the TGF-beta-binding site in alpha2M, five cDNA fragments, collectively encoding amino acids 122-1302, were expressed as glutathione S-transferase (GST) fusion proteins. In ligand blotting experiments, TGF-beta2 bound only to the fusion protein (**FP3**) that includes amino acids 614-797. **FP3** bound 125I-TGF-beta1 and 125I-TGF-beta2 in solution, preventing the binding of these growth factors to immobilized alpha2M-methylamine (alpha2M-MA). The IC50 values were 33 +/- 5 and 26 +/- 6 nM for TGF-beta1 and TGF-beta2, respectively; these values were comparable with or lower than those determined with native alpha2M or alpha2M-MA. A GST fusion protein that includes amino acids 798-1082 of alpha2M (**FP4**) and purified GST did not inhibit the binding of TGF-beta to immobilized alpha2M-MA. **FP3** (0.2 microM) neutralized the activity of TGF-beta1 and TGF-beta2 in fetal bovine heart endothelial (FBHE) cell proliferation assays; **FP4** was inactive in this assay. **FP3** also increased NO synthesis by RAW 264.7 cells, mimicking an alpha2M activity that has been attributed to the neutralization of endogenously synthesized TGF-beta. Thus, we have isolated a peptide corresponding to 13% of the alpha2M sequence that binds TGF-beta and neutralizes the activity of TGF-beta in two separate biological assays.

L12 ANSWER 12 OF 26 MEDLINE on STN
ACCESSION NUMBER: 1998316651 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9654079
TITLE: Transcriptional regulation of the non-specific cross-reacting antigen gene, a member of the carcinoembryonic antigen gene family up-regulated in colorectal carcinomas.
AUTHOR: Koops M D; Thompson J; Zimmermann W; Stanners C P
CORPORATE SOURCE: McGill Cancer Centre, McGill University, Montreal, Quebec, Canada.
SOURCE: European journal of biochemistry / FEBS, (1998 May 1) 253 (3) 778-86.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199807
ENTRY DATE: Entered STN: 19980811
Last Updated on STN: 19980811
Entered Medline: 19980729

AB Human non-specific cross-reacting antigen (NCA), a close relative of the tumor marker human carcinoembryonic antigen (CEA), is also an in vitro homotypic intercellular adhesion molecule capable of inhibiting differentiation when expressed ectopically by myoblasts.

Moreover, NCA appears to be overexpressed at the transcriptional level to a greater extent and more frequently in colorectal carcinomas than CEA. This study examines the transcriptional control mechanisms responsible for orchestrating NCA expression. The region within 284 bp upstream of the translational start site of the NCA gene was found to be capable of directing high levels of expression in functional promoter assays. Footprinting experiments identified three cis-acting elements and mobility-shift assays revealed that the first of these elements is bound by the upstream stimulating factors USF1 and USF2 while the other two are bound by the stimulatory proteins Sp1 and Sp3. No cis-acting elements corresponding to CEA footprint **FP4** or the silencer CEA **FP5** were detected in the NCA promoter, which may contribute to the differential expression of NCA versus CEA in tumorigenesis.

L12 ANSWER 13 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1997-012090 [01] WPIDS
DOC. NO. CPI: C1997-003401
TITLE: Isolated nucleic acid encoding malate permease and related vectors - used for increasing malate degradation during fermentation to eliminate excessive acidity in wine.
DERWENT CLASS: C06 D16
INVENTOR(S): GROBLER, J; JANSEN VAN VUUREN, H J; KRIZUS, A; OSOTHSILP-DE-EKNAMAKUL, C; PRETORIUS, I S; SUBDEN, R E; OSOTHSILP-DEEKNAMAKUL, C; VAN VUUREN, H J J; DE-EKNAMAKUL, C O
PATENT ASSIGNEE(S): (UYGU-N) UNIV GUELPH; (UYST-N) UNIV STELLENBOSCH; (GROB-I) GROBLER J; (KRIZ-I) KRIZUS A; (OSOT-I) OSOTHSILP-DE-EKNAMAKUL C; (PRET-I) PRETORIUS I S; (SUBD-I) SUBDEN R E; (VVUU-I) VAN VUUREN H J J
COUNTRY COUNT: 24
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9636715	A1	19961121	(199701)*	EN	80
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA HU JP US					
ZA 9603971	A	19970129	(199710)		99
AU 9656827	A	19961129	(199712)		
EP 827541	A1	19980311	(199814)	EN	
R: AT CH DE DK ES FR GR IT LI PT					
AU 718791	B	20000420	(200029)		
US 6274311	B1	20010814	(200148)		
US 2002081684	A1	20020627	(200245)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9636715	A1	WO 1996-CA320	19960517
ZA 9603971	A	ZA 1996-3971	19960517
AU 9656827	A	AU 1996-56827	19960517
EP 827541	A1	EP 1996-914819	19960517
		WO 1996-CA320	19960517

AU 718791	B		AU 1996-56827	19960517
US 6274311	B1		WO 1996-CA320	19960517
			US 1998-952365	19980528
US 2002081684	A1	Cont of	WO 1996-CA320	19960517
		Cont of	US 1998-952365	19980528
			US 2001-894993	20010628

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9656827	A	Based on	WO 9636715
EP 827541	A1	Based on	WO 9636715
AU 718791	B	Previous Publ.	AU 9656827
		Based on	WO 9636715
US 6274311	B1	Based on	WO 9636715
US 2002081684	A1	Cont of	US 6274311

PRIORITY APPLN. INFO: ZA 1995-4072 19950518
AN 1997-012090 [01] WPIDS
AB WO 9636715 A UPAB: 19970102
An isolated nucleic acid (I) encoding a protein (A) that mediates uptake of L-malate, succinate and malonate (collectively MSM), is new. Also new are: (1) isolated nucleic acid (Ia) encoding a fusion protein (FP) of (A) and a heterologous protein or peptide; (2) **recombinant** expression vectors containing (I) or (Ia); (3) host cell having (I) or (Ia) integrated into its genome; (4) isolated (A) and **FP**; (5) antibodies (Ab) against (A); and (6) method for identifying cpds. (B) that mediate transport of MSM.

USE - Host cells are used to produce **recombinant** (A). Cells transformed with (I) or (Ia) have increased uptake of MSM and are able to degrade malate, partic. that present in wine must during fermentation so as to remove excess acidity from the wine. Ab, opt. labelled, are used to detect (A) and probes derived from (I) are used to detect related genes. Sequences antisense to (I) can be used to reduce/inhibit uptake of MSM, e.g. in cases where the wine must contains insufficient acidity or where the yeast being used is partic. effective at degrading malate. Ab and (B) can be used similarly. (B) that stimulate (A) may also enhance malolactic or maloethanolic fermentation. (I) can also be incorporated into plants to improve energy efficiency.

ADVANTAGE - (I) is constitutively expressed and is not subject to catabolite repression as are known malate permeases. (A) provides a simple and relatively inexpensive way of removing malate, during or after fermentation.

L12 ANSWER 14 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1996-230603 [23] WPIDS
CROSS REFERENCE: 1994-217494 [26]; 1994-271898 [33]; 1994-341765 [42]; 1998-230234 [20]
DOC. NO. NON-CPI: N1996-193576
DOC. NO. CPI: C1996-072926
TITLE: Fusion proteins comprising non-toxin protein and part of toxin - useful to form anti-toxins against Clostridium botulinum type A, and C. difficile type toxins, and to treat C. difficile intoxication,

partic. diarrhoea.
DERWENT CLASS: B04 C06 D16 S03
INVENTOR(S): FIRCA, J R; KINK, J A; PADHYE, N V; STAFFORD, D C; THALLEY, B S; WILLIAMS, J A
PATENT ASSIGNEE(S): (OPHI-N) OPHIDIAN PHARM INC; (PROM-N) PROMEGA CORP; (ALLR) ALLERGAN BOTOX LTD; (ALLR) ALLERGAN INC
COUNTRY COUNT: 69
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9612802	A1	19960502	(199623)*	EN	336
RW: AT BE CH DE DK ES FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG					
W: AL AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UG US UZ VN					
AU 9539683	A	19960515	(199634)		
ZA 9508990	A	19960731	(199635)		433
NO 9701868	A	19970624	(199736)		
FI 9701732	A	19970623	(199738)		
EP 796326	A1	19970924	(199743)	EN	
R: AT BE CH DE DK ES FR GB GR IE IT LI LT LU LV MC NL PT SE SI					
BR 9509903	A	19971125	(199803)		
CZ 9701250	A3	19980318	(199817)		
US 5736139	A	19980407	(199821)		203
KR 97707285	A	19971201	(199847)		
US 5919665	A	19990706	(199933)		
HU 78048	T	19990728	(199936)		
AU 709586	B	19990902	(199948)		
NZ 295998	A	19991028	(199953)		
AU 9948763	A	19991125	(200006)		
MX 9702955	A1	19980701	(200012)		
AU 9963043	A	20000511	(200031)#		
EP 1041149	A2	20001004	(200051)	EN	
R: AT BE CH DE DK ES FR GB GR IE IT LI LT LU LV MC NL PT SE SI					
US 6290960	B1	20010918	(200157)		
CN 1176658	A	19980318	(200209)		
US 6365158	B1	20020402	(200226)		
JP 2002514886	W	20020521	(200236)		470
AU 747841	B	20020523	(200245)#		
NZ 337543	A	20020628	(200252)		
US 2003054493	A1	20030320	(200323)#		
AU 758820	B	20030403	(200335)#		
CA 2416318	A1	19960502	(200336)	EN	
US 6573003	B2	20030603	(200339)		
JP 2003137897	A	20030514	(200340)		51
US 6613329	B1	20030902	(200359)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9612802	A1	WO 1995-US13737	19951023
AU 9539683	A	AU 1995-39683	19951023
ZA 9508990	A	ZA 1995-8990	19951024

NO 9701868	A		WO 1995-US13737	19951023
			NO 1997-1868	19970423
FI 9701732	A		WO 1995-US13737	19951023
			FI 1997-1732	19970423
EP 796326	A1		EP 1995-937626	19951023
			WO 1995-US13737	19951023
BR 9509903	A		BR 1995-9903	19951023
			WO 1995-US13737	19951023
CZ 9701250	A3		WO 1995-US13737	19951023
			CZ 1997-1250	19951023
US 5736139	A	CIP of	US 1989-429791	19891031
		CIP of	US 1992-985321	19921204
		CIP of	US 1993-161907	19931202
		CIP of	US 1994-329154	19941024
		CIP of	US 1995-405496	19950316
		CIP of	US 1995-422711	19950414
			US 1995-480604	19950607
KR 97707285	A		WO 1995-US13737	19951023
			KR 1997-702691	19970424
US 5919665	A	CIP of	US 1989-429791	19891031
		CIP of	US 1992-985321	19921204
		CIP of	US 1993-161907	19931202
		CIP of	US 1994-329154	19941025
			US 1995-405496	19950316
HU 78048	T		WO 1995-US13737	19951023
			HU 1999-1238	19951023
AU 709586	B		AU 1995-39683	19951023
NZ 295998	A		NZ 1995-295998	19951023
			WO 1995-US13737	19951023
AU 9948763	A	Div ex	AU 1995-39683	19951023
			AU 1999-48763	19990916
MX 9702955	A1		MX 1997-2955	19970423
AU 9963043	A	Div ex	AU 1995-39683	19951023
			AU 1999-63043	19991202
EP 1041149	A2	Div ex	EP 1995-937626	19951023
			EP 2000-105371	19951023
US 6290960	B1	CIP of	US 1989-429791	19891031
		CIP of	US 1992-985321	19921204
		CIP of	US 1993-161907	19931202
		CIP of	US 1994-329154	19941024
		CIP of	US 1995-405496	19950316
		CIP of	US 1995-422711	19950414
		Cont of	US 1995-480604	19950607
			US 1997-915136	19970820
CN 1176658	A		CN 1995-196424	19951023
US 6365158	B1	CIP of	US 1989-429791	19891031
		CIP of	US 1992-985321	19921204
		CIP of	US 1993-161907	19931202
		Cont of	US 1994-329154	19941024
			US 1997-957310	19971023
JP 2002514886	W		WO 1995-US13737	19951023
			JP 1996-514127	19951023
AU 747841	B	Div ex	AU 1995-39683	19951023
			AU 1999-48763	19990916
NZ 337543	A		NZ 1995-337543	19951023
US 2003054493	A1	CIP of	US 1989-429791	19891031

Searcher : Shears 571-272-2528

		CIP of	US 1992-985321	19921204
		CIP of	US 1993-161907	19931202
		Cont of	US 1997-957310	19971023
			US 2001-11366	20011116
AU 758820	B	Div ex	AU 1995-39683	19951023
			AU 1999-63043	19991202
CA 2416318	A1	Div ex	CA 1995-2203504	19951023
			CA 1995-2416318	19951023
US 6573003	B2	CIP of	US 1989-429791	19891031
		CIP of	US 1992-985321	19921204
		CIP of	US 1993-161907	19931202
		Cont of	US 1994-329154	19941024
		Cont of	US 1997-957310	19971023
			US 2001-11366	20011116
JP 2003137897	A	Div ex	JP 1996-514127	19951023
			JP 2002-238940	19951023
US 6613329	B1	CIP of	US 1989-429791	19891031
		CIP of	US 1992-985321	19921204
		CIP of	US 1993-161907	19931202
		CIP of	US 1994-329154	19941024
		CIP of	US 1995-405496	19950316
		Cont of	US 1995-422711	19950414
			US 1998-84517	19980526

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9539683	A	Based on	WO 9612802
EP 796326	A1	Based on	WO 9612802
BR 9509903	A	Based on	WO 9612802
CZ 9701250	A3	Based on	WO 9612802
US 5736139	A	CIP of	US 5196193
		CIP of	US 5601823
KR 97707285	A	Based on	WO 9612802
US 5919665	A	CIP of	US 5196193
		CIP of	US 5601823
HU 78048	T	Based on	WO 9612802
AU 709586	B	Previous Publ.	AU 9539683
		Based on	WO 9612802
NZ 295998	A	Based on	WO 9612802
AU 9948763	A	Div ex	AU 709586
AU 9963043	A	Div ex	AU 709586
EP 1041149	A2	Div ex	EP 796326
US 6290960	B1	CIP of	US 5196193
		CIP of	US 5601823
		Cont of	US 5736139
		CIP of	US 5919665
US 6365158	B1	CIP of	US 5196193
		CIP of	US 5601823
JP 2002514886	W	Based on	WO 9612802
AU 747841	B	Div ex	AU 709586
		Previous Publ.	AU 9948763
NZ 337543	A	Div in	NZ 512238
US 2003054493	A1	CIP of	US 5196193
		CIP of	US 5601823

Searcher : Shears 571-272-2528

		Cont of	US 6365158
AU 758820	B	Div ex	AU 709586
		Previous Publ.	AU 9963043
US 6573003	B2	CIP of	US 5196193
		CIP of	US 5601823
		Cont of	US 6365158
US 6613329	B1	CIP of	US 5196193
		CIP of	US 5601823
		CIP of	US 5919665

PRIORITY APPLN. INFO: US 1995-480604 19950607; US 1994-329154 19941024; US 1995-405496 19950316; US 1995-422711 19950414; US 1989-429791 19891031; US 1992-985321 19921204; US 1993-161907 19931202; AU 1999-63043 19991202; US 1997-915136 19970820; US 1997-957310 19971023; US 2001-11366 20011116; US 1998-84517 19980526

AN 1996-230603 [23] WPIDS

CR 1994-217494 [26]; 1994-271898 [33]; 1994-341765 [42]; 1998-230234 [20]

AB WO 9612802 A UPAB: 20031128

Fusion protein (FP) comprising a polyhistidine tract and part of a toxin, is claimed. Also claimed are: (1) host cell containing a **recombinant** expression vector encoding a FP comprising a non-toxin protein sequence and part of the C. botulinum type A toxin sequence with the 1296 residue amino acid sequence given in the specification, where the cell expresses the toxin as a soluble protein at a level of at least 0.75% (pref. at least 20%) of the total cellular protein; (2) antibodies (Abs) raised by immunising a host with the **FP**; (3) compsn. comprising an avian neutralising antitoxin against part of Clostridium difficile toxin A and B; and (4) compsn. comprising an avian antitoxin against a clostridial toxin protein, in solid dosage form.

USE - The FPs are useful to generate neutralising antitoxin against C. botulinum type A, and C. difficile type toxins (claimed). The compositions are useful to treat symptoms of C. difficile intoxication, partic. diarrhoea. The Abs are used to detect C. difficile antigens in a sample, and to treat C. difficile related diarrhoea. They can also purify C. difficile toxins from a culture (claimed). The toxin fragments are useful in vaccines and diagnostic assays.

Dwg.0/58

L12 ANSWER 15 OF 26 MEDLINE on STN

ACCESSION NUMBER: 97038948 MEDLINE

DOCUMENT NUMBER: PubMed ID: 8884529

TITLE: Comparative epitope mapping of sera from United States (US) and Japanese patients with bullous pemphigoid (BP) to fusion proteins encoded by BPAG1.

AUTHOR: Rico M J; Hashimototo T; Watanabe K; Hall R P; Clark R B; Nishikawa T

CORPORATE SOURCE: Ronald O. Perelman Department of Dermatology, New York University, NY, USA.

CONTRACT NUMBER: KO8-ARO 1808 (NIAMS)

SOURCE: Journal of dermatological science, (1996 Sep) 12 (3)

238-45.
Journal code: 9011485. ISSN: 0923-1811.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970227
Last Updated on STN: 19970227
Entered Medline: 19970213

AB Sera from patients with bullous pemphigoid (BP) from the United States (US), Japan, and Britain demonstrate similar reactivity to the major target antigens BPAG1 and BPAG2. The purpose of the present study was to determine if the epitope specificity of circulating autoantibodies in patients with BP from the US and Japan is similar as mapped by binding to fusion proteins encoded by BPAG1. Sera from patients and controls with BP from the US and Japan were assayed for reactivity to intact BPAG1 and BPAG2 by immunoblot, and to fusion proteins encoded by BPAG1 by immunoblot and enzyme-linked immunosorbant assay (ELISA). Significant reactivity to fusion proteins encoded by the carboxyl region (FP 16-8) and coiled-coil region (**FP3**) was seen in sera from the US and Japanese patients, but not from normal controls from the US or Japan. Sera from US and Japanese patients differed in their response to **FP7**; namely, the reactivity of sera from US patients but not from Japanese patients to **FP7** was significantly different from the reactivity of their respective control sera. The reasons for this difference in reactivity are unknown but may reflect genetic or environmental factors relevant in the generation of an autoantibody response to these target antigens.

L12 ANSWER 16 OF 26 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 3
ACCESSION NUMBER: 1997:180561 BIOSIS
DOCUMENT NUMBER: PREV199799472274
TITLE: Genetic nature, stability, and improved virulence of hybrids from protoplast fusion in Beauveria.
AUTHOR(S): Couteaudier, Y.; Viaud, M.; Riba, G.
CORPORATE SOURCE: Station Recherches Lutte Biologique, INRA, La Miniere, 78285 Guyancourt Cedex, France
SOURCE: Microbial Ecology, (1996) Vol. 32, No. 1, pp. 1-10.
CODEN: MCBEBU. ISSN: 0095-3628.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Apr 1997
Last Updated on STN: 24 Apr 1997

AB Genetic improvement of two different strains of the entomopathogenic fungus Beauveria bassiana for more effective control of Ostrinia nubilalis and Leptinotarsa decemlineata was obtained by crosses with the insecticidal toxin-producing strain Beauveria sulfurescens. Protoplast fusion between diauxotrophic mutants resulted in the recovery of some stable prototrophic fusion products. The low levels of virulence of the wild type strain B. bassiana 28 isolated originally from L. decemlineata were enhanced both on L. decemlineata and O. nubilalis for one of the hybrids obtained (**FP 8**) from the cross B. bassiana 28 times B.

sulfurescens 2 Fusion product 25 obtained from the cross between B. sulfurescens and the highly pathogenic strain B. bassiana 147 showed a three-day reduction in the LT-50 towards O. nubilalis. Southern blot hybridization with nine probe-enzyme combinations were conducted on genomic DNAs from the original wild strains, parental mutant strains, and fusion products. Additive banding patterns or unique banding pattern of either parental strain was observed in five hybrids, indicating their status as **recombinant** and/or partially diploid. Combination of RFLP markers indicative of both parental genomes was never observed with fusion product FP 25. The stability of the virulence following passage through insect-host and stability of molecular structure for the fusion products **FP 8** and FP 25 suggest that asexual genetic recombination by protoplast fusion may provide an attractive method for the genetic improvement of biocontrol efficiency in entomopathogenic fungi.

L12 ANSWER 17 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1995-185778 [24] WPIDS
DOC. NO. CPI: C1995-086350
TITLE: New fusion protein for use in tumour therapy - comprising a tumour necrosis factor molecule and interleukin-1 beta residues 163-171..
DERWENT CLASS: B04 D16
INVENTOR(S): BOLLON, A P; SIDHU, R S
PATENT ASSIGNEE(S): (CYTO-N) CYTOCLONAL PHARM INC; (CYTO-N) CYTOCLONAL PHARMACEUTICS INC
COUNTRY COUNT: 57
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9512675	A1	19950511	(199524)*	EN	44
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ					
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK ES FI GB GE HU JP KE KG KP KR KZ LK LT LU LV MD MG MN MW NL NO NZ PL PT RO RU SD SE SI SK TJ TT UA US UZ VN					
AU 9511705	A	19950523	(199535)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9512675	A1	WO 1994-US12656	19941103
AU 9511705	A	AU 1995-11705	19941103

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9511705	A	Based on	WO 9512675

PRIORITY APPLN. INFO: US 1993-147673 19931104
AN 1995-185778 [24] WPIDS
AB WO 9512675 A UPAB: 19950626

A fusion protein (FP) is claimed comprising a tumour necrosis factor (TNF) molecule and an interleukin-1beta (IL-1beta) (residues 163-171) nonapeptide fused to the TNF.

Also claimed are: (1) a DNA sequence encoding the above FP; (2) a **recombinant** expression vector which expresses the above **FP**; (3) a process for prepare a FP comprising culturing a host cell comprising an expression vector that expresses a FP comprising TNF fused to IL-1beta.

USE - The FP can be used to enhance protection from irradiation and to accelerate the regeneration of haematopoietic progenitors in animals treated with cytotoxic anticancer drugs and irradiation. In partic. the FP can be used for the immunostimulation of a patient having undergone irradiation therapy (claimed). The FP can also selectively reduce the growth of cancer cells.

Dwg.0/3

L12 ANSWER 18 OF 26 MEDLINE on STN

ACCESSION NUMBER:	95315211 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 7794935
TITLE:	Characterization of the human glucocorticoid receptor promoter.
AUTHOR:	Nobukuni Y; Smith C L; Hager G L; Detera-Wadleigh S D
CORPORATE SOURCE:	Clinical Neurogenetics Branch, National Institute of Mental Health, National Institutes of Health, Bethesda, Maryland 20892, USA.
SOURCE:	Biochemistry, (1995 Jun 27) 34 (25) 8207-14. Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
OTHER SOURCE:	GENBANK-U10430
ENTRY MONTH:	199508
ENTRY DATE:	Entered STN: 19950817 Last Updated on STN: 20021218 Entered Medline: 19950801

AB To elucidate the functional elements that are involved in the regulation of the human glucocorticoid receptor (hGR) gene, transient expression, DNase I footprinting, and gel mobility shift analyses were conducted. We found that the hGR promoter region between -700 and +38 bp contained 11 footprinted sites. Deletion of the -374 to -183 bp region, which is highly conserved between human and mouse (93%), induced a 5-24-fold reduction in promoter activity in HeLa, NIH3T3, CV1, and HepG2 cells. Three footprints, **FP5**, **FP6**, and **FP7**, were shown to map to this region. In particular, the **FP7** site was found to be within the -374 to -347 bp region. Deletion of this region triggered a significant decline in promoter activity in HeLa and NIH3T3 cells but not in HepG2 cells. AP2 was found to bind **FP7**. In HepG2 cells AP2 elicited transactivation of the hGR promoter activity. Transfection data revealed that the upstream GC box-rich fragment between -700 and -375 bp induced a 4-7-fold activation of the heterologous tk promoter in an orientation-independent manner. Our studies demonstrate that several transcription factors are involved in regulating GR expression and that AP2 could function as an important positive

regulator of GR promoter activity.

L12 ANSWER 19 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1994-065704 [08] WPIDS
DOC. NO. CPI: C1994-029544
TITLE: Cloning and sequencing vector encoding toxic fusion protein - inactivated when foreign DNA is inserted, allowing direct selection of **recombinant** hosts.
DERWENT CLASS: B04 D16
INVENTOR(S): BERNARD, P; GABANT, P
PATENT ASSIGNEE(S): (ULBR) UNIV LIBRE BRUXELLES
COUNTRY COUNT: 45
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9403616	A2	19940217	(199408)*	FR	21
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL OA PT SE					
W: AU BB BG BR BY CA CZ FI HU JP KP KR KZ LK MG MN MW NO NZ PL RO RU SD SK UA US VN					
BE 1006085	A3	19940510	(199423)		19
AU 9345530	A	19940303	(199426)		
WO 9403616	A3	19940331	(199516)		
EP 652963	A1	19950517	(199524)	FR	
R: CH DE FR GB LI NL					
JP 08500484	W	19960123	(199642)		25
EP 652963	B1	19970924	(199743)	FR	13
R: CH DE FR GB LI NL					
DE 69314180	E	19971030	(199749)		
US 5910438	A	19990608	(199930)		
US 6180407	B1	20010130	(200108)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9403616	A2		WO 1993-BE51	19930802
BE 1006085	A3		BE 1992-696	19920731
AU 9345530	A		AU 1993-45530	19930802
WO 9403616	A3		WO 1993-BE51	19930802
EP 652963	A1		EP 1993-915577	19930802
			WO 1993-BE51	19930802
JP 08500484	W		WO 1993-BE51	19930802
			JP 1994-504840	19930802
EP 652963	B1		EP 1993-915577	19930802
			WO 1993-BE51	19930802
DE 69314180	E		DE 1993-614180	19930802
			EP 1993-915577	19930802
			WO 1993-BE51	19930802
US 5910438	A		WO 1993-BE51	19930802
			US 1995-379614	19950720
US 6180407	B1	CIP of	WO 1993-BE51	19930802
		CIP of	US 1995-379614	19950720
			US 1999-225152	19990104

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9345530	A	Based on	WO 9403616
EP 652963	A1	Based on	WO 9403616
JP 08500484	W	Based on	WO 9403616
EP 652963	B1	Based on	WO 9403616
DE 69314180	E	Based on	EP 652963
		Based on	WO 9403616
US 5910438	A	Based on	WO 9403616
US 6180407	B1	CIP of	US 5910438

PRIORITY APPLN. INFO: BE 1992-696 19920731
AN 1994-065704 [08] WPIDS
AB WO 9403616 A UPAB: 20010307
Cloning and/or sequencing vector comprises, in an autonomously replicable vector (2), at least one promoter (3) and at least one nucleotide sequence (4) encoding a toxic fusion protein (**FP**). (**4**) is made by fusing a sequence (5) having several cloning sites and a sequence (6) encoding a protein poison (I).

Also new are prokaryotic cells transfected with such a cloning vector and fragments of these vectors containing 10-30 nucleotides which hybridise with sequences on either side of (5).

USE/ADVANTAGE - The vectors are used for direct selection of **recombinant** clones and the fragments as primers for sequencing and/or amplification. The vectors can accommodate large nucleic acid fragments; are easily manipulated and can be propagated to high copy number without lethal effects on cells. Specific and reliable selection of **recombinants** is achieved and any foreign DNA fragment can be sequenced, amplified or characterised using the same primer. The foreign DNA is also easy to recover.
Dwg.0/4
ABEQ EP 652963 B UPAB: 19971030
Cloning and/or sequencing vector which is characterised in that it includes, incorporated into an autonomously replicating vector (2), at least one promoter nucleotide sequence (3) and at least one nucleotide sequence (4) which encodes a fusion protein which is active as a poison, the said nucleotide sequence (4) being obtained by fusing a coding nucleotide sequence (5), which comprises several unique cloning sites, and a nucleotide sequence (6) which encodes a protein poison.
Dwg.1/3

L12 ANSWER 20 OF 26 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1993-182493 [22] WPIDS
DOC. NO. CPI: C1993-080841
TITLE: Receptor protein comprising gp130 covalently linked to LIF receptor - binds to oncostatin M and LIF useful for treating Kaposis sarcoma, atherosclerosis, obesity etc..
DERWENT CLASS: B04 D16
INVENTOR(S): GEARING, D P
PATENT ASSIGNEE(S): (IMMV) IMMUNEX CORP
COUNTRY COUNT: 23
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9310151	A1	19930527	(199322)*	EN	79
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL SE					
W: AU CA FI JP KR NO					
AU 9332285	A	19930615	(199340)		
US 5262522	A	19931116	(199347)		40
JP 07501941	W	19950302	(199517)		
US 5426048	A	19950620	(199530)		45
EP 672061	A1	19950920	(199542)	EN	
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL SE					
EP 672061	A4	19951227	(199627)		
AU 670253	B	19960711	(199635)		
CA 2124085	C	19990511	(199937)	EN	

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9310151	A1		WO 1992-US10272	19921120
AU 9332285	A		AU 1993-32285	19921120
US 5262522	A		US 1991-797556	19911122
JP 07501941	W		WO 1992-US10272	19921120
			JP 1993-509585	19921120
US 5426048	A	Cont of	US 1991-797556	19911122
			US 1993-115370	19930831
EP 672061	A1		WO 1992-US10272	19921120
			EP 1993-900711	19921120
EP 672061	A4		EP 1993-900711	
AU 670253	B		AU 1993-32285	19921120
CA 2124085	C		CA 1992-2124085	19921120
			WO 1992-US10272	19921120

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9332285	A	Based on	WO 9310151
JP 07501941	W	Based on	WO 9310151
US 5426048	A	Cont of	US 5262522
EP 672061	A1	Based on	WO 9310151
AU 670253	B	Previous Publ.	AU 9332285
		Based on	WO 9310151
CA 2124085	C	Based on	WO 9310151

PRIORITY APPLN. INFO: US 1991-797556 19911122; US 1993-115370 19930831

AN 1993-182493 [22] WPIDS

AB WO 9310151 A UPAB: 19931115

A new receptor (R), able to bind oncostatin M (I) and leukaemia inhibitory factory (LIF), comprises a gp 130 polypeptide covalently coupled to LIF-receptor (LIF-R).

Also new are (1) isolated DNA sequences encoding (R), (2) **recombinant** expression vectors containing such DNA; (3) host cells containing these vectors; (4) fusion proteins (FP) containing an

antibody Fc region attached to the C-terminus of a soluble gp 130 polypeptide or of a soluble LIF-R polypeptide; (5) isolated DNA encoding **FP**; (6) homodimeric receptors consisting of two FP derived from LIF-R linked via disulphide bonds between the Fc fragments.

Pref. both components of (R) are soluble polypeptides and are connected by a polypeptide linker of 20-100 amino acids consisting of Gly, Asn, Ser, Thr or Ala.

USE/ADVANTAGE - (R) are useful for treating disorders mediated by (I) or LIF, e.g. atherosclerosis; obesity, disorders of bone or Ca metabolism; leukaemia; cachexia, etc. They have greater affinity for LIF and (I) than, respectively, LIF-R or gp 130 themselves. (R), or antibodies raised against them, may also be used to diagnose such disorders or for affinity purifcn. of gp 130 and LIF.

Dwg.0/7

ABEQ US 5262522 A UPAB: 19940111

Receptor binding oncostatin M and leukaemia inhibitory factor comprises gp130 covalently linked to LIF-R. gp130 is encoded by DNA encoding characteristics amino acid sequence SEQ ID NO:2 and DNA which can hybridise under moderately stringent conditions to the complement of the first DNA, and that biologically-active gp130 polypeptide is encoded. LIF-2 is encoded by DNA encoding characteristic amino acid sequence SEQ ID NO:6 and DNA which an hybridise under moderately strignent conditions to the complement of the other DNA, such that biologically active LIF-R polypeptide is encoded.

USE - Used for treating disorders mediated by either oncostatin M or LIF, e.g. Kaposis sarcinoma, athleroscleosis or obesity.

Dwg. 0/6

ABEQ US 5426048 A UPAB: 19950804

Isolated DNA encodes a **recombinant** fusion protein of Formula R1-L-R2 or R2-L-R1. R1 = gp. 130 polypeptide encoded by the DNA sequence given in the specification. R2 = LIF-R polypeptide encoded by the DNA sequence given in the specification. L = a polypeptide linker. Also claimed are a **recombinant** expression vector comprising the DNA a host cell contg. the vector and a process for preparing the fusion proteins.

USE - Used to produce fusion proteins which inhibit oncostatin M and leukemia inhibitory factor in the treatment of Kaposi's sarcoma, atherosclerosis, obesity, bone and calcium metabolism disorders, cachexia in AIDS patients etc. Also for diagnostic purposes.

Dwg.0/13

L12 ANSWER 21 OF 26 MEDLINE on STN
ACCESSION NUMBER: 94065233 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7504025
TITLE: IgG antibodies from patients with bullous pemphigoid bind to fusion proteins encoded by BPAg1 cDNA.
AUTHOR: Miller J E; Rico M J; Hall R P 3rd
CORPORATE SOURCE: Department of Medicine, Durham V.A. Medical Center, North Carolina.
CONTRACT NUMBER: 5K08-AR01808 (NIAMS) 5R01-AM-34718 (NIADDK)
SOURCE: Journal of investigative dermatology, (1993 Dec) 101 (6) 779-82.

Searcher : Shears 571-272-2528

Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199401
ENTRY DATE: Entered STN: 19940201
Last Updated on STN: 19960129
Entered Medline: 19940106

AB Bullous pemphigoid (BP) is an autoimmune blistering skin disease characterized in part by the presence of circulating and tissue-bound IgG antibodies directed against the epidermal basement membrane zone. IgG from over 95% of patients with BP have been shown to immunoprecipitate a 230-kD epidermal protein, BPAg1, which has been cloned and sequenced. Although sera from almost all patients with BP react with the 230-kD BP antigen the specific epitope(s) of BPAg1 that IgG binds is not known. We have generated fusion proteins from the 230-kD BP antigen cDNA and analyzed sera from patients with BP for binding to these fusion proteins by immunoblot. Sera from 21 of 30 (70%) patients with BP reacted with FP3A (amino acid 873-1193) compared to four of 13 (30%) normal subjects ($p < 0.02$). Sera from 10 of 30 (33%) patients reacted with **FP7** (AA1623-1812) and to **FP3** (AA1003-1193), compared to one of 22 (5%) and 0 of 19 (0%) controls, respectively. No significant reactivity was noted against two other fusion proteins (**FP6**, FP9). Twenty-four of 30 (80%) patients with BP reacted to at least one of three fusion proteins (**FP3**, FP3A, **FP7**) compared to three of 11 (27%) of the control subjects ($p < 0.003$). Fusion proteins **FP3**, FP3A, and **FP7** are at the amino- or carboxyl-terminal regions of the putative central alpha-helical coiled-coil rod domain of BPAg1, which has been postulated to be involved in the self-aggregation of BPAg1. These findings demonstrate that patients with bullous pemphigoid react with multiple regions of BPAg1 and suggest that part of the pathologic consequences of these auto-antibodies in patients with bullous pemphigoid may be by the disruption of the normal self-aggregation of the BPAg1.

L12 ANSWER 22 OF 26 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 92147859 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1737836
TITLE: Diagnostic value of a synthetic peptide derived from Echinococcus granulosus **recombinant** protein.
AUTHOR: Chamekh M; Gras-Masse H; Bossus M; Facon B; Dissous C; Tartar A; Capron A
CORPORATE SOURCE: Centre d'Immunologie et de Biologie Parasitaire, Unite Mixte INSERM U167-CNRS 624, Institut Pasteur, Lille, France.
SOURCE: Journal of clinical investigation, (1992 Feb) 89 (2) 458-64.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

OTHER SOURCE: GENBANK-M73719; GENBANK-M73720; GENBANK-M74034; GENBANK-M74320; GENBANK-M74321; GENBANK-M74322; GENBANK-M74323; GENBANK-M74324; GENBANK-S81163; GENBANK-S81167
ENTRY MONTH: 199203
ENTRY DATE: Entered STN: 19920405
Last Updated on STN: 19980206
Entered Medline: 19920318

AB A specific monoclonal antibody (MAb; EG 02 154/12) directed against a protein epitope of Echinococcus granulosus antigen 5 was used to screen a cDNA library constructed from E. granulosus protoscoleces RNA. One clone designated Eg14 was selected and shown to code for an amino acid sequence partially homologous to that of the clone Eg6 previously identified with the same MAb. Hydrophobic cluster analysis showed that both **recombinant** antigens may adopt a similar alpha-helical organization and share a common conformational epitope. A synthetic peptide (89-122) mimicking the conformational site of Eg6 and Eg14 was constructed and demonstrated to be able to inhibit binding of the MAb and human hydatid sera to the Eg6 fusion protein (**FP6**) or to native hydatid antigens. To assess the diagnostic value of the peptide 89-122, we tested sera from patients infected with different parasites for their antibody reactivity with this peptide in ELISA. A high binding sensitivity and specificity of IgG-A-M antibodies were obtained with E. granulosus-infected patient sera. Moreover, the peptide 89-122 was found to be specifically recognized by IgE antibodies from patients with hydatid disease. These results indicate the particular interest of this synthetic peptide as a standardized antigen in diagnosis and treatment surveillance of hydatidosis.

L12 ANSWER 23 OF 26 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 92005001 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1913346
TITLE: Cloning and characterization of the nifA gene from Herbaspirillum seropedicae strain Z78.
AUTHOR: Souza E M; Funayama S; Rigo L U; Pedrosa F O
CORPORATE SOURCE: Department of Biochemistry, Universidade Federal do Parana, Curitibia, PR, Brazil.
SOURCE: Canadian journal of microbiology, (1991 Jun) 37 (6) 425-9.
Journal code: 0372707. ISSN: 0008-4166.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199111
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911108

AB A genomic library of Herbaspirillum seropedicae was constructed and screened for the nifA gene by complementation of a nifA mutant of Azospirillum brasilense (**FP10**). A **recombinant** plasmid, pEMS1, capable of restoring acetylene reduction activity in the mutant **FP10**, was isolated and found to hybridize to the nifA gene of Klebsiella pneumoniae. The results suggest that nifA is involved in the regulation of nif genes in H. seropedicae.

L12 ANSWER 24 OF 26 MEDLINE on STN
ACCESSION NUMBER: 91246257 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1710034
TITLE: Molecular cloning of an Echinococcus granulosus protein expressing an immunogenic epitope of antigen 5.
AUTHOR: Facon B; Chamekh M; Dissous C; Capron A
CORPORATE SOURCE: Centre d'Immunologie et de Biologie Parasitaire, CNRS 624, Institut Pasteur, Lille, France.
SOURCE: Molecular and biochemical parasitology, (1991 Apr) 45 (2) 233-9.
Journal code: 8006324. ISSN: 0166-6851.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M55441; GENBANK-M58605; GENBANK-M64245; GENBANK-M90695; GENBANK-S78945; GENBANK-S78946; GENBANK-S78947; GENBANK-S78948; GENBANK-S78949; GENBANK-S78950
ENTRY MONTH: 199107
ENTRY DATE: Entered STN: 19910719
Last Updated on STN: 19960129
Entered Medline: 19910701

AB cDNA was synthesized from RNA extracted from Echinococcus granulosus protoscoleces and cloned in the lambda gt11 expression vector. A pool of 5 E. granulosus patient sera was used to screen the library and allowed the selection of 13 clones. Ten of these were shown to be identical, among which clone 6 (Eg6) was chosen for further analysis. The nucleotide sequence (456-bp) presented an entire open reading frame coding for 152 amino acids. The fusion protein (**FP6**) was recognized by a mouse monoclonal antibody (EG 02 154/12) specific for E. granulosus antigen 5. Moreover, the presence of antibodies to **FP6** seemed to be correlated to the ability of sera from hydatidosis patients to immunoprecipitate antigen 5. These results indicate that the cloned protein could be used as a standardized antigen for the diagnosis of hydatidosis.

L12 ANSWER 25 OF 26 MEDLINE on STN
ACCESSION NUMBER: 90217579 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1691240
TITLE: Production of rabbit antibodies against carboxy-terminal epitopes encoded by bullous pemphigoid cDNA.
AUTHOR: Tanaka T; Korman N J; Shimizu H; Eady R A; Klaus-Kovtun V; Cehrs K; Stanley J R
CORPORATE SOURCE: Dermatology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892.
SOURCE: Journal of investigative dermatology, (1990 May) 94 (5) 617-23.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199005
ENTRY DATE: Entered STN: 19900622
Last Updated on STN: 19960129
Entered Medline: 19900524

AB A partial cDNA clone (called BP cDNA) with coding sequences for the carboxy-terminal region of bullous pemphigoid (BP) antigen has been recently isolated and sequenced. In order to determine whether specific peptides encoded by the cDNA could be used to raise antibodies against BP antigen, fusion proteins derived from fragments of the BP cDNA and 17-mer or 19-mer synthetic peptides, corresponding to its deduced amino acid sequence, were used to generate rabbit antibodies. Three restriction enzyme fragments, 1179 bp (5' end), 264 bp (middle), and 546 bp (3' end), of the 1992 open reading frame (ORF) of BP cDNA were subcloned in frame into pEX plasmids to make beta-galactosidase fusion proteins FP1, FP2, and **FP3**, respectively. Fusion proteins of the predicted molecular weight, and which bound anti-beta-galactosidase antibodies, were produced, confirming the length of the predicted ORF. Rabbits immunized with FP1, but not **FP3**, produced antibodies, similar to authentic antibodies from BP patients, which: 1) bound the epidermal basement membrane at titers over 10,000, as determined by indirect immunofluorescence; 2) bound the basement membrane on the roof of 1 M NaCl-split skin; 3) immunoprecipitated the 230-kD BP antigen; and 4) bound the hemidesmosome, as determined by immunoelectron microscopy. Rabbits immunized with FP2 also produced lower titer BP-like antibodies. We further showed that short hydrophilic synthetic peptides, contained in FP1, could induce similar BP-like antibodies in rabbits at immunofluorescence titers up to 2560. These rabbit antibodies should prove useful for further studies on the function and structure of particular epitopes of BP antigen as well as on the pathophysiology of disease.

L12 ANSWER 26 OF 26 MEDLINE on STN
ACCESSION NUMBER: 89201244 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3071742
TITLE: Targeting a foreign protein to chloroplasts using fusions to the transit peptide of a chlorophyll a/b protein.
AUTHOR: Kavanagh T A; Jefferson R A; Bevan M W
CORPORATE SOURCE: Department of Molecular Genetics, Institute of Plant Science Research, Cambridge Laboratory, Trumpington, UK.
CONTRACT NUMBER: GM10798-02 (NIGMS)
SOURCE: Molecular & general genetics : MGG, (1988 Dec) 215 (1) 38-45.
Journal code: 0125036. ISSN: 0026-8925.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198905
ENTRY DATE: Entered STN: 19900306
Last Updated on STN: 19970203
Entered Medline: 19890519

AB We have constructed chimaeric genes consisting of sequences encoding

the transit peptide and 4, 16, 24, 53 or 126 amino-terminal residues of the mature chlorophyll a/b binding (Cab) apoprotein fused to the Escherichia coli gene encoding beta-glucuronidase (GUS). These genes were introduced into tobacco plants and the fate of the fusion proteins they encode was analysed. Less than 1% of the total activity of fusion proteins containing the transit peptide and 4 (**FP4**) or 16 (FP16) amino-terminal amino acids of the mature Cab protein was associated with chloroplasts. Moreover, **FP4** appears to be unprocessed. This is in striking contrast to fusion proteins containing the transit peptide and 24 (FP24), 53 (FP53) or 126 (FP126) amino-terminal residues of the mature Cab polypeptide. Approximately 98%, 96% or 75%, respectively, of the total activity of these fusion proteins was associated with purified intact chloroplasts, and protease protection experiments showed that of this, approximately 98%, 87% or 50%, respectively, was located within this organelle. Furthermore, both FP24 and FP53 appear to be processed. However, less than 10% of the activity of those fusion proteins translocated into chloroplasts was associated with thylakoid membranes.

FILE 'USPATFULL' ENTERED AT 09:03:08 ON 23 APR 2004
L13 87 S L8(S)RECOMBINAN?
L14 4 S L13(S)CRUZI

L14 ANSWER 1 OF 4 USPATFULL on STN
ACCESSION NUMBER: 2004:57966 USPATFULL
TITLE: Pharmaceutical compositions and treatment methods
INVENTOR(S): Ahlem, Clarence N., San Diego, CA, UNITED STATES
Heggie, William, Cabanas, PORTUGAL
Carvalho, Luis D., Paio Pires, PORTUGAL

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004043973	A1	20040304
APPLICATION INFO.:	US 2002-319356	A1	20021213 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-535675, filed on 23 Mar 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-190140P	20000316 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOLLIS-EDEN PHARMACEUTICALS, INC., 4435 EASTGATE MALL, SUITE 400, SAN DIEGO, CA, 92121
NUMBER OF CLAIMS: 53
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 9007
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides compositions comprising formula 1 steroids, e.g., 16α-bromo-3β-hydroxy-5α-androstan-17-one hemihydrate and one or more excipients, typically wherein the composition comprises less than about 3% water. The compositions are useful to make improved pharmaceutical formulations. The invention also provides methods of intermittent dosing of steroid

compounds such as analogs of 16α-bromo-3β-hydroxy-5α-androstan-17-one and compositions useful in such dosing regimens. The invention further provides compositions and methods to inhibit pathogen (viral) replication, ameliorate symptoms associated with immune dysregulation and to modulate immune responses in a subject using certain steroids and steroid analogs. The invention also provides methods to make and use these immunomodulatory compositions and formulations.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
INCL INCLM: 514/179.000
INCLS: 514/011.000; 514/045.000; 514/049.000; 552/633.000
NCL NCLM: 514/179.000
NCLS: 514/011.000; 514/045.000; 514/049.000; 552/633.000

L14 ANSWER 2 OF 4 USPATFULL on STN
ACCESSION NUMBER: 2003:332378 USPATFULL
TITLE: Pharmaceutical compositions and treatment methods
INVENTOR(S): Ahlem, Clarence Nathaniel, San Diego, CA, United States
de Carvalho, Luis Daniel dos Anjos, Paio Pires, PORTUGAL
Heggie, William, Palmela, PORTUGAL
PATENT ASSIGNEE(S): Hollis-Eden Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6667299	B1	20031223	
APPLICATION INFO.:	US 2000-535675		20000323	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-190140P	20000316	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Badio, Barbara P.
LEGAL REPRESENTATIVE: Muenchau, Daryl D.
NUMBER OF CLAIMS: 39
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 8994
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides compositions comprising, 16α-bromo-3β-hydroxy-5α-androstan-17-one hemihydrate and one or more excipients, typically wherein the composition comprises less than about 3% water. The compositions are useful to make improved pharmaceutical formulations. The invention also provides methods of intermittent dosing of steroid compounds such as analogs of 16α-bromo-3β-hydroxy-5α-androstan-17-one and compositions useful in such dosing regimens. The invention further provides compositions and methods to inhibit pathogen (viral) replication, ameliorate symptoms associated with immune dysregulation and to modulate immune responses in a subject using certain steroids and steroid analogs. The invention also provides methods to make and use these

immunomodulatory compositions and formulations.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
INCL INCLM: 514/178.000
INCLS: 552/536.000
NCL NCLM: 514/178.000
NCLS: 552/536.000

L14 ANSWER 3 OF 4 USPATFULL on STN
ACCESSION NUMBER: 2003:120747 USPATFULL
TITLE: Blood cell deficiency treatment method
INVENTOR(S): Ahlem, Clarence N., San Diego, CA, UNITED STATES
Reading, Christopher, San Diego, CA, UNITED STATES
Frincke, James, San Diego, CA, UNITED STATES
Stickney, Dwight, Granite Bay, CA, UNITED STATES
Lardy, Henry A., Madison, WI, UNITED STATES
Marwah, Padma, Middleton, WI, UNITED STATES
Marwah, Ashok, Middleton, WI, UNITED STATES
Prendergast, Patrick T., Straffan, IRELAND

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003083231	A1	20030501	
APPLICATION INFO.:	US 2002-87929	A1	20020301	(10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-675470, filed on 28 Sep 2000, PENDING
Continuation-in-part of Ser. No. US 2001-820483, filed on 29 Mar 2001, PENDING
Continuation-in-part of Ser. No. US 2000-535675, filed on 23 Mar 2000, PENDING
Continuation-in-part of Ser. No. US 1999-449004, filed on 24 Nov 1999, ABANDONED
Continuation-in-part of Ser. No. US 1999-449184, filed on 24 Nov 1999, ABANDONED
Continuation-in-part of Ser. No. US 1999-449042, filed on 24 Nov 1999, ABANDONED
Continuation-in-part of Ser. No. US 1999-461026, filed on 15 Dec 1999, ABANDONED
Continuation-in-part of Ser. No. US 2000-586673, filed on 1 Jun 2000, ABANDONED
Continuation-in-part of Ser. No. US 2000-586672, filed on 1 Jun 2000, ABANDONED
Continuation-in-part of Ser. No. US 1999-414905, filed on 8 Oct 1999, ABANDONED

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1999-161453P	19991025	(60)
	US 2001-272624P	20010301	(60)
	US 2001-323016P	20010911	(60)
	US 2001-340045P	20011130	(60)
	US 2001-328738P	20011011	(60)
	US 2001-338015P	20011108	(60)
	US 2001-343523P	20011220	(60)
	US 1999-126056P	19991019	(60)

Searcher : Shears 571-272-2528

	US 1999-124087P	19990311 (60)
	US 1998-109923P	19981124 (60)
	US 1998-109924P	19981124 (60)
	US 1998-110127P	19981127 (60)
	US 1998-112206P	19981215 (60)
	US 1999-145823P	19990727 (60)
	US 1999-137745P	19990603 (60)
	US 1999-140028P	19990616 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOLLIS-EDEN PHARMACEUTICALS, INC., 4435 EASTGATE MALL, SUITE 400, SAN DIEGO, CA, 92121
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
LINE COUNT: 19428
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to the use of compounds to treat a number of conditions, such as thrombocytopenia, neutropenia or the delayed effects of radiation therapy. Compounds that can be used in the invention include methyl-2,3,4-trihydroxy-1-O-(7,17-dioxoandrost-5-ene-3β-yl)-β-D-glucopyranosiduronate, 16α,3α-dihydroxy-5α-androstan-17-one or 3,7,16,17-tetrahydroxyandrost-5-ene, 3,7,16,17-tetrahydroxyandrost-4-ene,3,7,16,17-tetrahydroxyandrost-1-ene or 3,7,16,17-tetrahydroxyandrostane that can be used in the treatment method.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
INCL INCLM: 514/002.000
INCLS: 514/063.000; 514/026.000; 514/044.000; 514/169.000; 514/173.000
NCL NCLM: 514/002.000
NCLS: 514/063.000; 514/026.000; 514/044.000; 514/169.000; 514/173.000

L14 ANSWER 4 OF 4 USPATFULL on STN
ACCESSION NUMBER: 2003:86817 USPATFULL
TITLE: Immune modulation method using steroid compounds
INVENTOR(S): Ahlem, Clarence N., San Diego, CA, UNITED STATES
Frincke, James M., San Diego, CA, UNITED STATES
dos Anjos de Carvalho, Luis Daniel, Paio Pires, PORTUGAL
Heggie, William, Palmela, PORTUGAL
Prendergast, Patrick T., County Kildare, IRELAND
Reading, Christopher L., San Diego, CA, UNITED STATES
Thadikonda, Krupakar Paul, Gaithersburg, MD, UNITED STATES
Vernon, Russell N., Oak Hills, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003060425	A1	20030327
APPLICATION INFO.:	US 2001-820483	A1	20010329 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-449184, filed on 24 Nov 1999, ABANDONED
Continuation-in-part of Ser. No. US 1999-414905,

filed on 8 Oct 1999, ABANDONED
Continuation-in-part of Ser. No. US 1999-449004, filed on 24 Nov 1999, ABANDONED
Continuation-in-part of Ser. No. US 2000-535675, filed on 23 Mar 2000, PENDING
Continuation-in-part of Ser. No. US 1999-449042, filed on 24 Nov 1999, ABANDONED
Continuation-in-part of Ser. No. US 2000-675470, filed on 28 Sep 2000, PENDING
Continuation-in-part of Ser. No. US 2000-586673, filed on 1 Jun 2000, ABANDONED
Continuation-in-part of Ser. No. US 2000-586672, filed on 1 Jun 2000, ABANDONED
Continuation-in-part of Ser. No. US 1999-461026, filed on 15 Dec 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-109924P	19981124 (60)
	US 1999-140028P	19990616 (60)
	US 1998-109923P	19981124 (60)
	US 1999-126056P	19991019 (60)
	US 1999-124087P	19990311 (60)
	US 1998-110127P	19981127 (60)
	US 1999-161453P	19991025 (60)
	US 1999-145823P	19990727 (60)
	US 1999-137745P	19990603 (60)
	US 1998-112206P	19981215 (60)
	US 2000-257071P	20001220 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOLLIS-EDEN PHARMACEUTICALS, INC., 4435 EASTGATE MALL, SUITE 400, SAN DIEGO, CA, 92121
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 14708
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides compositions comprising formula 1 steroids, e.g., 16α-bromo-3 β-hydroxy-5α-androstan-17-one hemihydrate and one or more excipients, including compositions that comprise a liquid formulation comprising less than about 3% v/v water. The compositions are useful to make improved pharmaceutical formulations. The invention also provides methods of intermittent dosing of steroid compounds such as analogs of 16α-bromo-3β-hydroxy-5α-androstan-17-one and compositions useful in such dosing regimens. The invention further provides compositions and methods to inhibit pathogen replication, ameliorate symptoms associated with immune dysregulation and to modulate immune responses in a subject using the compounds. The invention also provides methods to make and use these immunomodulatory compositions and formulations.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
INCL INCLM: 514/026.000
INCLS: 514/044.000; 514/063.000; 514/099.000; 514/169.000;

514/173.000
NCL NCLM: 514/026.000
NCLS: 514/044.000; 514/063.000; 514/099.000; 514/169.000; 514/173.000

(FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO, USPATFULL' ENTERED AT 09:04:23 ON 23 APR 2004)

L15 364 SEA ABB=ON PLU=ON "KIRCHHOFF L"?/AU
L16 1751 SEA ABB=ON PLU=ON "OTSU K"?/AU - Author(s)
L17 37 SEA ABB=ON PLU=ON L15 AND L16
L18 0 SEA ABB=ON PLU=ON (L15 OR L16) AND L8
L19 272 SEA ABB=ON PLU=ON (L15 OR L16) AND CRUZI
L20 23 SEA ABB=ON PLU=ON L19 AND RECOMBINAN?
L21 15 SEA ABB=ON PLU=ON L20 AND (DIAGNOS? OR DETERM? OR DET## OR SCREEN? OR DETECT?)
L22 46 SEA ABB=ON PLU=ON L17 OR L21
L23 16 DUP REM L22 (30 DUPLICATES REMOVED)

L23 ANSWER 1 OF 16 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 1

ACCESSION NUMBER: 2001:484271 BIOSIS
DOCUMENT NUMBER: PREV200100484271
TITLE: Polypeptides for diagnosing infection with trypanosoma cruzi.
AUTHOR(S): **Kirchhoff, Louis V.** [Inventor, Reprint author]; **Otsu, Keiko** [Inventor]
CORPORATE SOURCE: 204 Lexington Ave., Iowa City, IA, 52246-2413, USA
PATENT INFORMATION: US 6228601 May 08, 2001
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (May 8, 2001) Vol. 1246, No. 2. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Oct 2001
Last Updated on STN: 23 Feb 2002

AB Polypeptides are disclosed that are useful for diagnosing American trypanosomiasis, or Chagas disease, a disease caused by the infectious agent Trypanosoma cruzi. The polypeptides have a sequence that corresponds to the amino acid sequence of at least one of the C-terminal and N-terminal nonrepetitive regions of TCR27 protein. The polypeptide additionally may comprise an amino acid sequence of one or more repeats from the central region of TCR27 protein. In a preferred embodiment, the polypeptide corresponds to the N-terminal nonrepetitive region of TCR27 protein and at least one repeat from the central region of TCR27 protein, and does not correspond to the C-terminal nonrepetitive region. The polypeptides may further comprise a linker sequence at either the N-terminus or the C-terminus to facilitate attachment or conjugation to a carrier molecule in a liquid or solid support system for use in a sensitive assay for detecting T. cruzi infection.

L23 ANSWER 2 OF 16 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2001:55958 BIOSIS
DOCUMENT NUMBER: PREV200100055958

TITLE: Evidence of Trypanosoma **cruzi** infection (Chagas' disease) among patients undergoing cardiac surgery.

AUTHOR(S): Leiby, David A. [Reprint author]; Rentas, Francisco J.; Nelson, Kenrad E.; Stambolis, Veronica A.; Ness, Paul M.; Parnis, Cheryl; McAllister, Hugh A., Jr.; Yawn, David H.; Stumpf, Robert J.; **Kirchhoff, Louis V.**

CORPORATE SOURCE: Transmissible Diseases Department, American Red Cross, 15601 Crabbs Branch Way, Rockville, MD, 20855, USA
leibyd@usa.redcross.org

SOURCE: Circulation, (December 12, 2000) Vol. 102, No. 24, pp. 2978-2982. print.
CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jan 2001
Last Updated on STN: 12 Feb 2002

AB Background: Trypanosoma **cruzi**, the agent of Chagas' heart disease, is transmitted by triatomine insects and by blood transfusion. The emigration of several million people from T **cruzi**-endemic countries to the United States has raised concerns regarding a possible increase in cases of Chagas' heart disease here, as well as an increased risk of transfusion-transmitted T **cruzi**. To investigate these 2 possible outcomes, we tested a repository of blood specimens from multiply transfused cardiac surgery patients for antibodies to T **cruzi**. Methods and Results: Postoperative blood specimens from 11 430 cardiac surgery patients were tested by enzyme immunoassay, and if repeat-reactive, were confirmed by radioimmunoprecipitation. Six postoperative specimens (0.05%) were confirmed positive. Corresponding preoperative specimens, available for 4 of these patients, were also positive. The other 2 patients had undergone heart transplantations. Tissue samples from their excised hearts were tested for T **cruzi** by polymerase chain reaction and were positive. Despite the fact that several of these 6 patients had histories and clinical findings suggestive of Chagas' disease, none of them were **diagnosed** with or tested for it. Patient demographics showed that 5 of 6 positive patients were Hispanic, and overall, 2.7% of Hispanic patients in the repository were positive. Conclusions: No evidence for transfusion-transmitted T **cruzi** was found. All 6 seropositive patients apparently were infected with T **cruzi** before surgery; however, a **diagnosis** of Chagas' disease was not known or even considered in any of these patients. Indeed, Chagas' disease may be an underdiagnosed cause of cardiac disease in the United States, particularly among patients born in countries in which T **cruzi** is endemic.

L23 ANSWER 3 OF 16 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2001:43542 BIOSIS
DOCUMENT NUMBER: PREV200100043542

TITLE: Prevalence of American trypanosomiasis (Chagas disease) among dogs in Oklahoma.

AUTHOR(S): Bradley, Kristy K. [Reprint author]; Bergman, Douglas K.; Woods, J. Paul; Crutcher, James M. [Reprint author]; **Kirchhoff, Louis V.**
CORPORATE SOURCE: Acute Disease Service, Oklahoma State Department of Health, 1000 NE 10th St, Oklahoma City, OK, 73117-1299, Canada
SOURCE: Journal of the American Veterinary Medical Association, (December 15, 2000) Vol. 217, No. 12, pp. 1853-1857. print.
CODEN: JAVMA4. ISSN: 0003-1488.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Jan 2001
Last Updated on STN: 12 Feb 2002

AB Objective-To **determine** the prevalence of Trypanosoma **cruzi** infection among dogs in Oklahoma.
Design-Cross-sectional study. Animals-301 owned or impounded dogs related by ownership or general geographic location to 3 dogs **determined** to have trypanosomiasis. Procedures-Blood samples were obtained from dogs between November 1996 and September 1997. Infection status was **determined** by use of a radioimmunoprecipitation assay. Second blood samples were obtained from some of the seropositive dogs for study by hemoculture and polymerase chain reaction (PCR) assay. Sites where infected dogs were found were inspected for triatomine insects, and light traps were used for vector trapping. Results-11 (3.6%) dogs were seropositive for T **cruzi** infection. Ten of the 11 were owned rural hunting dogs. Protozoal organisms isolated from the blood of 1 seropositive dog were identified as T **cruzi** by PCR testing. Only 1 adult Triatoma sanguisuga was captured in a light trap at a site near infected dogs; this insect was not infected. Conclusions and Clinical Relevance-Our findings suggest that T **cruzi** is enzootic in eastern Oklahoma. Measures that would reduce the risk of dogs acquiring T **cruzi** infection are unlikely to be acceptable to their owners, and no effective drugs are available for treatment. The presence of T **cruzi**-infected dogs poses a threat of transmission to persons at risk of exposure to contaminated blood Veterinarians who practice in the southern United States should be cognizant of this blood borne zoonosis and educate all personnel about appropriate precautions.

L23 ANSWER 4 OF 16 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 2
ACCESSION NUMBER: 1999:153345 BIOSIS
DOCUMENT NUMBER: PREV199900153345
TITLE: Polypeptides for diagnosing infection with Trypanosoma cruzi.
AUTHOR(S): **Kirchhoff, L. V** [Inventor]; **Otsu, K.** [Inventor]
CORPORATE SOURCE: 204 Lexington Ave., Iowa City, Iowa 52246-2413, USA
PATENT INFORMATION: US 5876734 March 2, 1999
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (March 2, 1999) Vol. 1220, No. 1, pp. 419. print.
CODEN: OGUPE7. ISSN: 0098-1133.

DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Apr 1999
Last Updated on STN: 16 Apr 1999

L23 ANSWER 5 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 1999:60584 HCAPLUS
DOCUMENT NUMBER: 130:321327
TITLE: Expression of a marker for intracellular Trypanosoma cruzi amastigotes in extracellular spheromastigotes
AUTHOR(S): Teixeira, Santuza M. R.; **Otsu, Keiko**; Hill, Kent L.; **Kirchhoff, Louis V.**; Donelson, John E.
CORPORATE SOURCE: Department of Biochemistry, University of Iowa, Iowa, IA, 52242, USA
SOURCE: Molecular and Biochemical Parasitology (1999), 98(2), 265-270
CODEN: MBIPDP; ISSN: 0166-6851
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A green fluorescent protein (GFP)-expressing marker under control of Trypanosoma cruzi (Tc) rRNA, joined immediately upstream to an Tc amastin gene 5'-UTR and downstream to an amastin gene 3'-UTR plus intergenic region, was inserted into a plasmid pTCR17-2::Neo, in which the coding region for the 14-amino acid repeats of cytoskeletal protein TCR was replaced with the coding region for neomycin phosphotransferase. Elevated expression in spheromastigotes of GFP under the control of an amastigote-specific up-regulatory sequence indicates that extracellular spheromastigotes are equivalent to intracellular amastigotes at the gene expression level.
REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 6 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 1996:201380 HCAPLUS
DOCUMENT NUMBER: 124:280613
TITLE: Maxicircle genomic organization and editing of an ATPase subunit 6 RNA in Trypanosoma cruzi
AUTHOR(S): Ochs, Diane E.; **Otsu, Keiko**; Teixeira, Santuza M. R.; Moser, David R.; **Kirchhoff, Louis V.**
CORPORATE SOURCE: Recombinant DNA Core Facility, University of Iowa, Iowa City, Iowa, USA
SOURCE: Molecular and Biochemical Parasitology (1996), 76(1,2), 267-78
CODEN: MBIPDP; ISSN: 0166-6851
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The DNA sequence of a 5736-nucleotide (nt) Trypanosoma cruzi maxicircle fragment was determined Sequence comparisons indicate that its 5' terminus is the homolog of the downstream portion of the NADH

dehydrogenase subunit 7 gene and that its 3' region is homologous to the maxicircle unidentified reading frame II gene. The region between these two gene segments contains six addnl. genes that encode mitochondrial proteins, including ATPase subunit 6 (A6). Comparison of the A6 maxicircle DNA sequence with that of an A6 cDNA indicates that the A6 RNA is extensively edited throughout its length. A 49-nt sequence that could serve as template for transcription of a guide RNA for editing a segment of the A6 RNA was found in one of 24 minicircle variable regions sequenced. Moreover, the presence of an RNA having this sequence was demonstrated in an RNAse protection assay. This is the first identification of a guide RNA template in a T. cruzi minicircle. Taken together, our findings suggest that T. cruzi and Trypanosoma brucei brucei are phylogenetically closer to each other than they are to Leishmania tarentolae, despite the relative similarity of the life cycles of the latter and T. cruzi.

L23 ANSWER 7 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 1995:963702 HCAPLUS
DOCUMENT NUMBER: 123:337440
TITLE: Polypeptides of antigen TCR27 for diagnosing infection with Trypanosoma cruzi
INVENTOR(S): **Kirchhoff, Louis V.; Otsu, Keiko**
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9525797	A1	19950928	WO 1995-US3191	19950320
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, UZ				
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5876734	A	19990302	US 1994-216894	19940324
CA 2186235	AA	19950928	CA 1995-2186235	19950320
AU 9519982	A1	19951009	AU 1995-19982	19950320
BR 9507182	A	19971014	BR 1995-7182	19950320
US 6228601	B1	20010508	US 1998-115746	19980715

PRIORITY APPLN. INFO.: US 1994-216894 A 19940324
WO 1995-US3191 W 19950320

AB Polypeptides are disclosed that are useful for diagnosing American Trypanosomiasis, or Chagas disease, a disease caused by the infectious agent Trypanosoma cruzi. The polypeptides have a sequence that corresponds to the amino acid sequence of at least one of the C-terminal and N-terminal non-repetitive regions of TCR27 protein. The polypeptide addnl. may comprise an amino acid sequence of one or more repeats from the central region of TCR27 protein. In

a preferred embodiment, the polypeptide corresponds to the N-terminal non-repetitive region of TCR27 protein and at least one repeat from the central region of TCR27 protein, and does not correspond to the C-terminal non-repetitive region. The polypeptides may further comprise a linker sequence at either the N-terminus or the C-terminus to facilitate attachment or conjugation to a carrier mol. in a liquid or solid support system for use in a sensitive assay for detecting T. cruzi infection.

L23 ANSWER 8 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 1996:118234 HCAPLUS
DOCUMENT NUMBER: 124:196637
TITLE: Trypanosoma cruzi: interruption of both alleles of a gene encoding a protein containing 14-amino-acid repeats by targeted insertion of NEOr and HYGr
AUTHOR(S): **Otsu, Keiko**; Donelson, John E.; **Kirchhoff, Louis V.**
CORPORATE SOURCE: Department of Internal Medicine, University of Iowa, Iowa City, IA, 52242, USA
SOURCE: Experimental Parasitology (1995), 81(4), 529-35
CODEN: EXPAAA; ISSN: 0014-4894
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In Trypanosoma cruzi approx. 90% of the 121- and 176-kDa cytoskeletal proteins encoded by the two alleles of the TCR27 gene is composed of 14-amino-acid repeats. To gain insight into the function of the TCR27 proteins we replaced the corresponding regions of 42-nucleotide repeats in the two alleles with the NEOr and HYGr genes. Analyses of DNAs and RNAs from four clones resistant to both G418, a neomycin analog, and hygromycin showed that in both cases the repetitive regions had in fact been deleted. In addition, the absence of expression of the 14-amino-acid repeats was confirmed in Western blots. In axenic cultures growth rates of the morphol. unchanged, doubly resistant organisms were not different from those of wild-type parasites. However, the doubly resistant organisms proliferated more slowly in cultured mammalian cells than did wild-type parasites. These findings indicate that the absence of the TCR27 repetitive regions is detrimental, but not fatal, to the parasites.

L23 ANSWER 9 OF 16 MEDLINE on STN
ACCESSION NUMBER: 94075782 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8254156
TITLE: Chagas disease. American trypanosomiasis.
AUTHOR: **Kirchhoff L V**
CORPORATE SOURCE: Department of Internal Medicine, University of Iowa, Iowa City.
SOURCE: Infectious disease clinics of North America, (1993 Sep) 7 (3) 487-502. Ref: 93
Journal code: 8804508. ISSN: 0891-5520.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

Searcher : Shears 571-272-2528

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199401
ENTRY DATE: Entered STN: 19940203
Last Updated on STN: 19940203
Entered Medline: 19940107

AB Chagas disease, caused by the protozoan parasite, Trypanosoma **cruzi**, is a major source of morbidity and death in Latin America. Many infected immigrants from that region now reside in the United States, posing a risk of transfusion-associated transmission of the organism. Serologic testing is the cornerstone of **diagnosing** chronic T. **cruzi** infections, and improved assays are needed. Drug treatment is problematic because the two available drugs can have severe side effects and lack efficacy. T. **cruzi** infection can be particularly severe in immunosuppressed patients.

L23 ANSWER 10 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 1993:141018 HCAPLUS
DOCUMENT NUMBER: 118:141018
TITLE: Interruption of a Trypanosoma cruzi gene encoding a protein containing 14-amino acid repeats by targeted insertion of the neomycin phosphotransferase gene
AUTHOR(S): **Otsu, Keiko**; Donelson, John E.; **Kirchhoff, Louis V.**
CORPORATE SOURCE: Dep. Intern. Med., Univ. Iowa, Iowa City, IA, USA
SOURCE: Molecular and Biochemical Parasitology (1993), 57(2), 317-30
CODEN: MBIPDP; ISSN: 0166-6851
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In T. cruzi, the cause of Chagas' disease in Latin America, a large proportion of the antigenic proteins described to date have repetitive domains. In earlier work the authors identified a partial length cDNA, designated TCR27, encoding approx. 26 copies of a 14-amino acid repeat and a unique 61-amino acid C-terminal region. The goal of the current project was to replace the repetitive region of a TCR27 gene with the neomycin phosphotransferase gene (NEOr). A pBluescript-based vector was constructed in which the 0.9-kb NEOr coding region replaced the 2.9-kb internal repetitive segment of a TCR27 gene and was inframe with its nonrepetitive 5' coding sequence (pTCR27-2::NEO). Epimastigotes were electroporated in the presence of linearized pTCR27-2::NEO and transfected clones were selected on solid medium containing G418. Southern and Northern analyses of DNAs and RNAs from 4 G418-resistant clones showed that in all cases the repetitive region in the smaller of the 2 TCR27 genes (TCR27-2) had been replaced by NEOr. The absence of the native TCR27-2 protein in the transfected clones was confirmed by Western blot. In axenic cultures growth rates of epimastigotes bearing an interrupted TCR27-2 gene were not different from those of wild-type parasites. In addition, there was no relative impairment of the four transfected clones' ability to proliferate in cultured mammalian cells. The fact that the clones having the interrupted TCR27-2 gene were not impaired biol. suggests that the length of the repetitive region of

the TCR27 protein is not a critical factor for survival.

L23 ANSWER 11 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 8
ACCESSION NUMBER: 1992:103569 HCAPLUS
DOCUMENT NUMBER: 116:103569
TITLE: Recombinant Leishmania Hsp90 and Hsp70 are recognized by sera from visceral leishmaniasis patients but not Chagas' disease patients
AUTHOR(S): De Andrade, Cynthia R.; **Kirchhoff, Louis V.**; Donelson, John E.; **Otsu, Keiko**
CORPORATE SOURCE: Dep. Intern. Med., Univ. Iowa, Iowa City, IA, 52242, USA
SOURCE: Journal of Clinical Microbiology (1992), 30(2), 330-5
CODEN: JCMIDW; ISSN: 0095-1137
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Approx. 70% of the cDNA clones identified by immunoscreening Leishmania donovani expression libraries with blood serum from a patient with visceral leishmaniasis (kala-azar) encoded the highly conserved Hsp90 and Hsp70 members of the heat shock protein family. Recombinant fusion proteins containing the C-terminal portions of L. donovani Hsp90 and Hsp70, were used as target antigens in ELISA of various sera. Sera from 4 patients with visceral leishmaniasis recognized the recombinant Leishmania Hsp90 and Hsp70, while sera from 7 patients with Chagas disease did not, despite the fact that Trypanosoma cruzi Hsp90 and Hsp70 share >80% amino acid identity with their counterparts in Leishmania spp. Thus, Leishmania Hsp90 and Hsp70 elicit strong humoral responses and are potential candidates for specific serodiagnostic assays capable of distinguishing between L. donovani and T. cruzi infections.

L23 ANSWER 12 OF 16 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1993:177706 BIOSIS
DOCUMENT NUMBER: PREV199344085306
TITLE: Stable integrative transformation of Trypanosoma cruzi with the bacterial NEO-r gene: Interruption of a gene encoding a protein containing 14-amino acid repeats.
AUTHOR(S): **Otsu, K.** [Reprint author]; Donelson, J. E.; **Kirchhoff, L. V.**
CORPORATE SOURCE: Dep. Intern. Med., Univ. Iowa, Howard Hughes Med. Inst., Iowa City, IA 52242, USA
SOURCE: Memorias do Instituto Oswaldo Cruz Rio de Janeiro, (1992) Vol. 87, No. SUPPL. 2, pp. 135.
Meeting Info.: XIX Annual Meeting on Basic Research in Chagas' Disease. Caxambu, Brazil. November 11-13, 1992.
CODEN: MIOCAS. ISSN: 0074-0276.
DOCUMENT TYPE: Conference; (Meeting)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Apr 1993
Last Updated on STN: 2 Apr 1993

L23 ANSWER 13 OF 16 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on

STN
ACCESSION NUMBER: 1991:150774 BIOSIS
DOCUMENT NUMBER: PREV199140070379; BR40:70379
TITLE: USE OF **RECOMBINANT** LEISHMANIA-DONOVANI HSP-LIKE POLYPEPTIDES IN THE **DIAGNOSIS** OF VISCERAL LEISHMANIASIS.
AUTHOR(S): ANDRADE C R [Reprint author]; ANDRADE P P; **KIRCHHOFF L V**; DONELSON J E
CORPORATE SOURCE: DEP HISTOL AND EMBRIOL, UNIV FEDERAL DE PERNAMBUCO, RECIFE, PE, BRAZIL
SOURCE: Memorias do Instituto Oswaldo Cruz, (1990) Vol. 85, No. SUPPL. 1, pp. 92.
Meeting Info.: PROCEEDINGS OF THE XVII ANNUAL MEETING ON BASIC RESEARCH IN CHAGAS' DISEASE, CAXAMBU, MINAS GERAIS, BRAZIL, NOVEMBER 5-6, 1990. MEM INST OSWALDO CRUZ RIO J.
CODEN: MIOCAS. ISSN: 0074-0276.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 23 Mar 1991
Last Updated on STN: 23 Mar 1991

L23 ANSWER 14 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 9
ACCESSION NUMBER: 1989:495079 HCAPLUS
DOCUMENT NUMBER: 111:95079
TITLE: Trypanosoma cruzi expresses diverse repetitive protein antigens
AUTHOR(S): Hoft, Daniel F.; Kim, Kwang S.; **Otsu, Keiko**; Moser, David R.; Yost, W. John; Blumin, Joel H.; Donelson, John E.; **Kirchhoff, Louis V.**
CORPORATE SOURCE: Dep. Intern. Med., Univ. Iowa, Iowa City, IA, 52242, USA
SOURCE: Infection and Immunity (1989), 57(7), 1959-67
CODEN: INFIBR; ISSN: 0019-9567
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A T. cruzi cDNA expression library was screened with human and rabbit anti-T. cruzi sera and cDNA clones were identified that encode polypeptides containing tandemly arranged repeats which are 6-34 amino acids in length. The peptide repeats encoded by these cDNAs varied markedly in sequence, copy number, and location relative to the polyadenylation site of the mRNAs from which they were derived. The repeats were specific for T. cruzi, but in each case the sizes of the corresponding mRNAs and the total number of repeat copies encoded varied considerably among different isolates of the parasite. Expression of the peptide repeats was not stage specific. One of the peptide repeats occurred in a protein with mol. weight (Mr) of >200,000 and one was in a protein of Mr 75,000-105,000. The frequent occurrence and diversity of these peptide repeats suggested that they may play a role in the ability of the parasite to evade immune destruction in its invertebrate and mammalian hosts, but the primary roles of these macromols. may be unrelated to the host-parasite relationship.

L23 ANSWER 15 OF 16 MEDLINE on STN
ACCESSION NUMBER: 88315072 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2457589
TITLE: Ubiquitin genes in trypanosomatidae.
AUTHOR: **Kirchhoff L V**; Kim K S; Engman D M; Donelson J E
CORPORATE SOURCE: Department of Internal Medicine, University of Iowa, Iowa City 52242.
CONTRACT NUMBER: AI18954 (NIAID)
AI24711 (NIAID)
GM07337 (NIGMS)
SOURCE: Journal of biological chemistry, (1988 Sep 5) 263 (25) 12698-704.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-J03945
ENTRY MONTH: 198809
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19880928

AB A ubiquitin encoding cDNA from Trypanosoma **cruzi**, the protozoan cause of Chagas' disease, was isolated by immunoscreening a lambda gt11 expression library with serum from a mouse chronically infected with this parasite. The cDNA encodes a precursor protein consisting of four tandem repeats of ubiquitin differing from that of Saccharomyces cerevisiae in four positions, followed by an unrelated 52-amino acid tail containing a putative metal and nucleic acid binding domain. Southern and Northern blots of DNA and RNA from various strains of T. **cruzi** and from several African trypanosome and Leishmania isolates revealed dramatic differences in the numbers and sizes of ubiquitin genes and transcripts. One of the T. **cruzi** isolates examined has as many as 10 ubiquitin genes, while a strain of Leishmania donovani appears to have only 1. Forty or more tandemly arranged ubiquitin coding repeats are present in some genes, while others have only two or three. Evidence for stage-specific expression of a ubiquitin transcript was found in one strain, but no stress-induced changes in the pattern of transcripts were **detected** in the one isolate examined. Thus the cellular requirements for ubiquitin in trypanosomatids can be supplied by diversely organized genes containing highly variable numbers of ubiquitin coding repeats.

L23 ANSWER 16 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 10
ACCESSION NUMBER: 1986:18434 HCAPLUS
DOCUMENT NUMBER: 104:18434
TITLE: Molecular mimicry of a carbohydrate epitope on a major surface glycoprotein of Trypanosoma **cruzi** by using anti-idiotypic antibodies
AUTHOR(S): Sacks, David L.; **Kirchhoff, Louis V.**; Hieny, Sara; Sher, Alan
CORPORATE SOURCE: Lab. Parasit. Dis., Natl. Inst. Allergy Infect. Dis., Bethesda, MD, 20892, USA
SOURCE: Journal of Immunology (1985), 135(6), 4155-9

CODEN: JOIMA3; ISSN: 0022-1767
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The use of anti-idiotypic antibodies (Ab2) to induce antimicrobial immunity might be particularly advantageous with respect to responses directed against carbohydrate **determinants**, because it may not be feasible to reproduce these epitopes by **recombinant** DNA technol. In the present studies, rabbit Ab2 were produced against a recurrent BALB/c idiotype defined by a monoclonal antibody (WIC 29.26) with specificity for a carbohydrate epitope of a major surface glycoprotein of T. **cruzi**. The Ab2 induced specific antibodies in mice, rabbits, and guinea pigs, and reacted with parasite-induced anti-T. **cruzi** antibodies from mice and rabbits as well as humans. The behavior of these Ab2 is therefore consistent with that of the antigen itself and suggests that mol. mimicry of carbohydrate epitopes can be easily achieved.

FILE 'HOME' ENTERED AT 09:10:11 ON 23 APR 2004

FILE 'REGISTRY' ENTERED AT 09:23:26 ON 23 APR 2004
E FP9/CN 5

-key terms

FILE 'HCAPLUS' ENTERED AT 09:23:33 ON 23 APR 2004
L24 22 S FP9 OR FP 9
L25 3 S L24 AND RECOMBINAN?
L26 3 S L25 NOT (L9 OR L4)

L26 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 23 Feb 2004
ACCESSION NUMBER: 2004:144882 HCAPLUS
DOCUMENT NUMBER: 140:197796
TITLE: Enhanced CD8+ T Cell Immune Responses and Protection Elicited against Plasmodium berghei Malaria by Prime Boost Immunization Regimens Using a Novel Attenuated Fowlpox Virus
AUTHOR(S): Anderson, Richard J.; Hannan, Carolyn M.; Gilbert, Sarah C.; Laidlaw, Stephen M.; Sheu, Eric G.; Korten, Simone; Sinden, Robert; Butcher, Geoffrey A.; Skinner, Michael A.; Hill, Adrian V. S.
CORPORATE SOURCE: Wellcome Trust Centre for Human Genetics, University of Oxford, Oxford, UK
SOURCE: Journal of Immunology (2004), 172(5), 3094-3100
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Sterile immunity can be provided against the pre-erythrocytic stages of malaria by IFN-γ-secreting CD8+ T cells that recognize parasite-infected hepatocytes. In this study, we have investigated the use of attenuated fowlpox virus (FPV) strains as **recombinant** vaccine vectors for eliciting CD8+ T cells against Plasmodium berghei. The gene encoding the P. berghei circumsporozoite (PbCS) protein was inserted into an FPV vaccine strain licensed for use in chickens, Webster's FPV, and the novel FPV vaccine strain **FP9** by homologous recombination. The novel **FP9** strain proved more potent as a vaccine for eliciting CD8+ T cell responses against the PbCS Ag. Sequential immunization with rFP9 and **recombinant** modified vaccinia virus Anakara (MVA) encoding the PbCS protein, administered by clin. acceptable routes, elicited potent CD8+ T cell responses against the PbCS protein. This immunization regimen elicited substantial protection against a stringent liver-stage challenge with P. berghei and was more immunogenic and protective than DNA/MVA prime/boost immunization. However, further improvement was not achieved by sequential (triple) immunization with a DNA vaccine, **FP9**, and MVA.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
ED Entered STN: 16 Jan 2004
ACCESSION NUMBER: 2004:39255 HCAPLUS
DOCUMENT NUMBER: 140:109758

TITLE: A Plasmodium falciparum candidate vaccine based on a six-antigen polyprotein encoded by **recombinant** poxviruses

AUTHOR(S): Prieur, Eric; Gilbert, Sarah C.; Schneider, Joerg; Moore, Anne C.; Sheu, Eric G.; Goonetilleke, Nilu; Robson, Kathryn J. H.; Hill, Adrian V. S.

CORPORATE SOURCE: Weatherall Institute of Molecular Medicine and Cellular Immunology and Vaccine Development Group, Nuffield Department of Medicine, John Radcliffe Hospital, University of Oxford, Oxford, OX3 9DU, UK

SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2004), 101(1), 290-295
CODEN: PNASA6; ISSN: 0027-8424

PUBLISHER: National Academy of Sciences

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To generate broadly protective T cell responses more similar to those acquired after vaccination with radiation-attenuated Plasmodium falciparum sporozoites, the authors have constructed candidate subunit malaria vaccines expressing six preerythrocytic antigens linked together to produce a 3,240-aa-long polyprotein (L3SEPTL). This polyprotein was expressed by a plasmid DNA vaccine vector (DNA) and by two attenuated poxvirus vectors, modified vaccinia virus Ankara (MVA) and fowlpox virus of the **FP9** strain. MVA-L3SEPTL boosted anti-thrombospondin-related adhesive protein (anti-TRAP) and anti-liver stage antigen 1 (anti-LSA1) CD8+ T cell responses when primed by single antigen TRAP- or LSA1-expressing DNAs, resp., but not by DNA-L3SEPTL. However, prime boost regimes involving two heterologous viral vectors expressing L3SEPTL induced a strong cellular response directed against an LSA1 peptide located in the C-terminal region of the polyprotein. Peptide-specific T cells secreted IFN-γ and were cytotoxic. IFN-γ-secreting T cells specific for each of the six antigens were induced after vaccination with L3SEPTL, supporting the use of polyprotein inserts to induce multispecific T cells against P. falciparum. The use of polyprotein constructs in nonreplicating poxviruses should broaden the target antigen range of vaccine-induced immunity and increase the number of potential epitopes available for immunogenetically diverse human populations.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN

ED Entered STN: 06 Aug 1998

ACCESSION NUMBER: 1998:489967 HCAPLUS

DOCUMENT NUMBER: 129:255686

TITLE: **Recombinant** fowlpox virus expressing newcastle disease virus haemagglutinin-neutramindase protein and its protective effect

AUTHOR(S): Cao, Dianjun; Liu, Huili; Wang, Lilin; Min, Ping; Zhang, Ying; Lu, Jingliang

CORPORATE SOURCE: State Laboratory of Veterinary Biotechnology,

Harbin Veterinary Research Institute, CAAS, Harbin, 150001, Peop. Rep. China
SOURCE: Zhongguo Mianyixue Zazhi (1998), 14(1), 61-64
CODEN: ZMZAEE; ISSN: 1000-484X
PUBLISHER: Zhongguo Mianyixue Zazhi Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB A **recombinant** fowlpox virus transfer vector was constructed by cloning haemagglutinin-Neuraminidase (HN) cDNA gene of Newcastle disease virus strain F 48E9 under control of P7.5 promoter. The vector was contransfected into chicken embryo cells with **FP9**. Pos. **recombinants** were selected by three cycles of plaque purification and Southern blot. Chicken embryo cells transfected with **recombinants** expressed two kinds of HN-sized proteins (MW 74 kDa; MW 68 kDa) by immunofluorescence and Western blot. Both of them were HN-specific proteins and could react with anti-HN antibody. Their mol. weight were coincided with glycosylated and unglycosylated NDV HN proteins. Cells transfected with **recombinant** rFPV-HN could adsorb chicken red blood cells and the cells surfaces exhibited neuraminidase activity. Both of these activities could be inhibited by anti-HN antibody. Four groups of SPF chickens were inoculated with the cells infected with the **recombinant** virus, La Sota and **FP9** resp. Chickens inoculated with the **recombinant** virus developed antibodies against NDV. The antibody titers could protect chickens against the NDV F48E9 challenge though they were not as high as that of chickens inoculated with NDV La Sota.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 09:24:48 ON 23 APR 2004)
L27 10 S L25
L28 9 S L27 NOT L11
L29 3 DUP REM L28 (6 DUPLICATES REMOVED)

L29 ANSWER 1 OF 3 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2004099014 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 14978115
TITLE: Enhanced CD8+ T cell immune responses and protection elicited against Plasmodium berghei malaria by prime boost immunization regimens using a novel attenuated fowlpox virus..
AUTHOR: Anderson Richard J; Hannan Carolyn M; Gilbert Sarah C; Laidlaw Stephen M; Sheu Eric G; Korten Simone; Sinden Robert; Butcher Geoffrey A; Skinner Michael A; Hill Adrian V S
CORPORATE SOURCE: Wellcome Trust Centre for Human Genetics, University of Oxford, Oxford, United Kingdom.. randerson@oxxonpharmaccines.com
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (2004 Mar 1) 172 (5) 3094-100.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Abridged Index Medicus Journals; Priority Journals

ENTRY DATE: Entered STN: 20040302
Last Updated on STN: 20040302

AB Sterile immunity can be provided against the pre-erythrocytic stages of malaria by IFN-gamma-secreting CD8(+) T cells that recognize parasite-infected hepatocytes. In this study, we have investigated the use of attenuated fowlpox virus (FPV) strains as **recombinant** vaccine vectors for eliciting CD8(+) T cells against Plasmodium berghei. The gene encoding the P. berghei circumsporozoite (PbCS) protein was inserted into an FPV vaccine strain licensed for use in chickens, Webster's FPV, and the novel FPV vaccine strain **FP9** by homologous recombination. The novel **FP9** strain proved more potent as a vaccine for eliciting CD8(+) T cell responses against the PbCS Ag. Sequential immunization with rFP9 and **recombinant** modified vaccinia virus Anakara (MVA) encoding the PbCS protein, administered by clinically acceptable routes, elicited potent CD8(+) T cell responses against the PbCS protein. This immunization regimen elicited substantial protection against a stringent liver-stage challenge with P. berghei and was more immunogenic and protective than DNA/MVA prime/boost immunization. However, further improvement was not achieved by sequential (triple) immunization with a DNA vaccine, **FP9**, and MVA.

L29 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2004013079 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14694197
TITLE: A Plasmodium falciparum candidate vaccine based on a six-antigen polyprotein encoded by **recombinant** poxviruses.
AUTHOR: Prieur Eric; Gilbert Sarah C; Schneider Joerg; Moore Anne C; Sheu Eric G; Goonetilleke Nilu; Robson Kathryn J H; Hill Adrian V S
CORPORATE SOURCE: Weatherall Institute of Molecular Medicine and Cellular Immunology, Nuffield Department of Medicine, University of Oxford, John Radcliffe Hospital, Oxford OX3 9DU, United Kingdom.. eric.prieur@well.ox.ac.uk
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (2004 Jan 6) 101 (1) 290-5.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 20040108
Last Updated on STN: 20040302
Entered Medline: 20040225

AB To generate broadly protective T cell responses more similar to those acquired after vaccination with radiation-attenuated Plasmodium falciparum sporozoites, we have constructed candidate subunit malaria vaccines expressing six preerythrocytic antigens linked together to produce a 3240-aa-long polyprotein (L3SEPTL). This polyprotein was expressed by a plasmid DNA vaccine vector (DNA) and by two attenuated poxvirus vectors, modified vaccinia virus Ankara (MVA) and fowlpox virus of the **FP9** strain.

MVAL3SEPTL boosted anti-thrombospondin-related adhesive protein (anti-TRAP) and anti-liver stage antigen 1 (anti-LSA1) CD8(+) T cell responses when primed by single antigen TRAP- or LSA1-expressing DNAs, respectively, but not by DNA-L3SEPTL. However, prime boost regimes involving two heterologous viral vectors expressing L3SEPTL induced a strong cellular response directed against an LSA1 peptide located in the C-terminal region of the polyprotein. Peptide-specific T cells secreted IFN-gamma and were cytotoxic. IFN-gamma-secreting T cells specific for each of the six antigens were induced after vaccination with L3SEPTL, supporting the use of polyprotein inserts to induce multispecific T cells against P. falciparum. The use of polyprotein constructs in nonreplicating poxviruses should broaden the target antigen range of vaccine-induced immunity and increase the number of potential epitopes available for immunogenetically diverse human populations.

L29 ANSWER 3 OF 3 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 2003-513700 [48] WPIDS
DOC. NO. CPI: C2003-137608
TITLE: Treating and/or preventing e.g. malaria or tuberculosis, or eliciting an immune response, comprises administering a priming composition and a boosting composition containing a non-replicating viral vector in either order.
DERWENT CLASS: B04 D16
INVENTOR(S): ANDERSON, R; GILBERT, S; HILL, A; LAIDLAW, S; SKINNER, M
PATENT ASSIGNEE(S): (ISIS-N) ISIS INNOVATION LTD
COUNTRY COUNT: 102
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003047617	A2	20030612	(200348)*	EN	95
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
GB 2382578	A	20030604	(200349)		
AU 2002347317	A1	20030617	(200419)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003047617	A2	WO 2002-GB5411	20021202
GB 2382578	A	GB 2001-28733	20011130
AU 2002347317	A1	AU 2002-347317	20021202

FILING DETAILS:

PATENT NO	KIND	PATENT NO

AU 2002347317 A1 Based on WO 2003047617

PRIORITY APPLN. INFO: US 2001-334649P 20011130; GB 2001-28733 20011130

AN 2003-513700 [48] WPIDS

AB WO2003047617 A UPAB: 20030729

NOVELTY - Treating and/or preventing a disease in a subject comprising administering two compositions, each containing a non-replicating viral vector, is new. At least one of the compositions comprises a poxvirus vector derived from a fowlpox virus.

DETAILED DESCRIPTION - Treating and/or preventing a disease in a subject comprises administering:

(a) a composition containing a first non-replicating viral vector; and

(b) a composition containing a second non-replicating viral vector; and optionally

(c) a composition comprising a DNA vaccine or a third non-replicating viral vector.

At least one of the compositions comprises a poxvirus vector derived from a fowlpox virus.

INDEPENDENT CLAIMS are also included for the following:

(1) eliciting an immune response or boosting a pre-existing immune response in a subject by administering a composition comprising a non-replicating viral vector, where the vector is or is derived from **FP9**;

(2) a fowlpox virus genome having a modified form of one or more wild-type FPV genes selected from FPV001, FPV018, FPV054, FPV063, FPV066, FPV070, FPV071, FPV093, FPV097, FPV098, FPV115, FPV124, FPV125, FPV127, FPV158, FPV159, FPV160, FPV190, FPV191, FPV207, FPV219, FPV220, FPV221, FPV222, FPV239, FPV241, FPV242, FPV243, FPV244, FPV245, FPV246, FPV247, and FPV260;

(3) a viral particle comprising a genome of (2);

(4) a priming or boosting agent comprising a viral particle of (3);

(5) a vaccination kit comprising a first composition containing a fowlpox viral particle, and a second composition containing a second non-replicating viral vector for simultaneous, separate or sequential administration; and

(6) a boosting composition comprising a non-replicating viral vector capable of boosting a pre-existing immune response in a primate subject.

ACTIVITY - Virucide; Tuberculostatic; Protozoacide; Antipyretic; Cytostatic; Hepatotropic; Antibacterial.

MECHANISM OF ACTION - Vaccine.

Female Balb/c mice were immunized intravenously with 1 multiply 106 plaque forming units (pfu) of **FP9** or FPV-M alone or expressing Plasmodium berghei CSP (PbCSP). Seven days following immunization, T-cell immune response elicited in splenocytes was determined using the IFN gamma ELSPOT assay. The response against the **recombinant** antigen, PbCSP, was determined using the MHC-class I restricted Pb9 epitope. As a positive control, the T-cell response against whole virus was determined by exposing immune splenocytes to those infected with fowlpox virus. Results show that the response elicited by **recombinant FP9** was significantly higher than that elicited by the DNA-vaccine,

although lower than that elicited by **recombinant** MVA. FP9PbCSP boosted the CD asterisk + T cell immune response primed by DNA vaccination, as well as acting as either a priming or boosting agent in combination with MVAPbCSP. Priming with FP9PbCSP and subsequent boosting with MVAPbCSP induced considerably higher levels of protection against challenge with P. berghei sporozoites than other homologous or heterologous prime-boost immunization regimes.

USE - The non-replicating viral vector is useful in a vaccine for an animal, particularly mammal such as a primate, specifically human. The priming or boosting composition, or the kit is useful for manufacturing a medicament for treating and/or preventing a disease which is or results from a chronic infection such as malaria, tuberculosis or East Coast fever, or for eliciting a T-cell immune response in a subject. Non-cultured CEF cells are useful for growing an avipox virus, such as fowlpox virus. (All claimed.) The method or the vaccine may further be used to treat or prevent influenza, hepatitis, human papilloma virus and other viral infections, malignancies such as tumors, leishmaniasis, listeriosis, and theileria.

Dwg.0/18

FILE 'USPATFULL' ENTERED AT 09:25:25 ON 23 APR 2004

L30 6 S L24(S)RECOMBINAN?

L31 0 S L30(S)CRUZI

FILE 'HOME' ENTERED AT 09:26:01 ON 23 APR 2004